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SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

I.—HOW TO OBVIATE FAILURES IN THE RESULTS OF TONSILLECTOMY.*

DR. DAVID H. JONES, New York.

I can best illustrate my remarks by demonstration of lantern slides. (Demonstration of slides showing form of history card and physical examination, operative and postoperative records kept at the Manhattan Eye, Ear and Throat Hospital. Also, demonstration of slides showing statistical data on tonsillectomies.)

You have seen the history card and the methods used to record the results both before and after operation, and will now relate the various complications which have been noted. I wish it understood that no criticism is directed against any person and trust that these remarks will be accepted in the spirit given.

Everything has been done to lessen postoperative complications, but the human element enters into this work; some

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, Jan. 17, 1934.

operators do excellent work; others, no matter how much effort is expended, never succeed.

Hemorrhage is the most frequent complication. Formerly if 40 operations were performed in an afternoon, it was expected that there would be from 15 to 18 hemorrhages. Now 15 to 18 hemorrhages a month call for explanations.

Adenoid hemorrhages alone occur, and 98 per cent were checked by removal of a piece of adenoid.

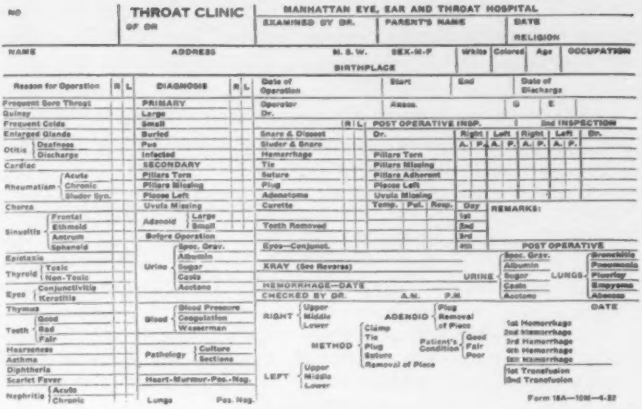
One of the first questions usually asked is: How many deaths have we had from hemorrhage? The answer is, none. All patients with severe hemorrhages are given one or more blood transfusions, and we know that this has been the main factor in preventing deaths from hemorrhage.

Primary hemorrhages occurring in the first 24 hours are much easier to control than secondary hemorrhages where one sees only an ooze in the tonsil fossa; no vessels to clamp and tie. A tourniquet to the extremities for one hour has been a great help in this condition. One patient had a hemorrhage 11 days after operation (the longest on our records).

One boy, age 11 years, said to be a hemophiliac whose coagulation time while under treatment with calcium lactate over a period of five months ranged from 12 to four minutes, bled so much at the time of operation that it was necessary to give five blood transfusions (5,210 cc.). Another patient, whose hemoglobin was as low as 32 per cent, red cells 1,600,000, received 6,210 cc.

One rather rare complication occurred, namely, removal of the epiglottis by a very good surgeon, and it happened when the infra tonsil of French was being removed in a colored child. We endeavored to determine the cause, and our observations show that the epiglottis in all colored children extends much farther forward on the tongue than in white children. The anesthesia was given by a substitute anesthetist, and the patient was not fully anesthetized, gagging reflex being partially present, consequently making it more difficult for the surgeon. Patient remained under observation for a week, was laryngoscoped, showing a complete amputation of the epiglottis, from which she suffered no ill effects.

Following operation two patients had perversion of taste and one loss of taste.



New York, 19

I, the undersigned, herewith give my consent to the Surgeons of the Manhattan Eye, Ear and Throat Hospital to perform such operations that in their judgment may be necessary, in the case of

Signed

Witness

RE-EXAMINED

BY DATE

1. IS PARENT SATISFIED
2. DOES CHILD HAVE SORE THROAT
3. IS CHILD MOUTH BREATHER
4. DOES CHILD HAVE FREQUENT COLDS
5. HAS CHILD GAINED WEIGHT LBS.
6. WHAT COMPLICATIONS:
   OTITIS { DEAFNESS { R. L. / DISCHARGE { R. L.
7. PILLARS TORN
8. PILLARS MISSING
9. PILLARS ADHERENT
10. PIECES LEFT
11. UVULA MISSING
12. SIZE OF GLANDS { LARGE / MEDIUM / NORMAL
13. IMPROVEMENT
14. IMPROVEMENT (Rheumatic Condition)

REMARKS:

XRAY EXAMINATION

Chart 1.

One patient with a foreign body in the lung, a tooth was dislodged at time of operation, and after two unsuccessful bronchoscopies the patient coughed up the tooth. Since then

when it is even suspected that a tooth is missing or if an instrument breaks, the patient is X-rayed immediately, report being furnished in 15 minutes.

NOTICE TO FEMALE PATIENTS.

Do not accept a date for tonsil operation which falls within the five days previous to your monthly period or the five days during this period.

Chart 2.

INSTRUCTIONS FOR THE CARE AND TREATMENT OF PATIENTS FOLLOWING OPERATION FOR REMOVAL OF ADENOIDS AND TONSILS.

PATIENTS MUST BE KEPT IN BED FOR THREE DAYS after operation, then three days in the house. After this patient may be outdoors at mid-day if weather is fair.

BOWELS MUST MOVE FREELY AT LEAST ONCE DAILY. Give both ADULTS and CHILDREN milk of magnesia the first thing in the morning.

DIET: Fluids the first 48 hours, ice cream, cornstarch, custard, junket and milk, then well cooked cereal, soups, milk toast and poached egg. DRINK WATER ABUNDANTLY.

DO NOT COUGH, HAWK OR CLEAR THROAT AS THIS MAY START BLEEDING.

Parents are requested to bring their children back for examination ONE WEEK AFTER OPERATION on the day written on your clinic card at 1 P.M., except Saturday.

All ADULT patients are requested to return as above.

IF ANY BLEEDING FROM THROAT SHOULD OCCUR RETURN TO THE HOSPITAL AT ONCE.

IF ANY SYMPTOMS OF COUGH DEVELOP WITHIN TEN DAYS AFTER OPERATION, COME TO THE HOSPITAL.

KEEP THIS SLIP AND YOUR CLINIC CARD, and bring the child again in three months on the day printed on your clinic card at 1 P.M., except Saturday. The doctor would like to see what benefits have been secured, and whether the condition at that time is satisfactory.

Chart 3.

Out of 52,467 patients operated on, there were five deaths. First, an adult from anesthesia given by an interne; second, a child from heat prostration; following this death, no child under 5 years of age has been operated on after three days of excessive humidity. Third, a child, age 20 months, with enlarged cervical glands, whose parents were advised to

postpone operation until glands had become smaller, came with a note from family physician asking for removal of adenoids and tonsils. Operation was performed and at its conclusion child collapsed and death followed immediately; cause of death, status lymphaticus (X-ray after death); no autopsy performed. Fourth death, a child, septicemia with metastatic pyogenic abscesses. Terminal bronchopneumonia. Fifth, young adult male, who at the time of operation had seven ties on the right and six on the left side, the operation taking 45 minutes, and as soon as patient reacted from the anesthetic began to bleed from adenoid and was brought back

REASONS FOR OPERATION — YEAR 1933.

Age	Rheumatism	Cardiac
1-10	13	29
10-16	24	15
16-25	137	40
25-35	198	48
35-45	176	31
45-55	66	13
Total	614	176

Chart 4.

to operating room, where five plugs were inserted and one in left fossa. At 6 P.M., patient was again taken to operating room, where three sutures were placed in each tonsil fossa, 500 cc. normal saline given, the following day owing to complete atelectasis of left lung patient was bronchoscoped and 200 cc. of fluid aspirated, temperature dropped and respirations easier. Following day, evidence of left lobar pneumonia seen by X-ray and physical examination; 750 cc. saline and glucose given, 750 cc. blood transfusion, and death occurred on fourth day. It was then learned from his relatives that he was a hemophiliac, but he did not so inform us.

A number of pulmonary complications, tracheobronchitis, bronchitis, broncho- and lobar pneumonia, have occurred. All patients are now being de-etherized following operation, and so far these conditions are practically eliminated. Formerly it was said that a considerable number of patients developed lung abscess after being operated on at Manhattan, but have

only been able to find records of six for the years 1931 and 1933.

OPERATIONS ACCORDING TO AGE — YEAR 1933.

	1-10		10-16		16-25		25-35		35-45		45-55		Pillars	P's Left
	Gen.	Loc.	Gen.	Loc.	Gen.	Loc.	Gen.	Loc.	Gen.	Loc.	Gen.		T & M	
Opt.	6138	2	1665	103	1151	579	413	358	74	96	14			
Total	6138		1667		1254		992		432		110		1106	194
Hem.	230		99		89		77		37		16			

Hemorrhages.

Adenoid hemorrhage	Local	General	Total
115	133	415	548

OPERATIONS.

	Loc.	Gen.	Total	Hem.	Post-opt.	S & D	Sluder	Ties	Operations	Hem.
1930									May 1, 1929	
Nov. &									Jan. 1, 1934	
Dec.	1399	10526	11925	474	9246					
1931									Pri.	Sec.
1932	1087	10914	12001	579	10320					
1933	1187	9396	10583	548	9394					
										3¼% 1¼%
Total	3673	30836	34509	1601	28960	10080	24429	55725	52467	

HEMORRHAGE RECORD BOOK KEPT IN EACH WARD.

Name		Address		Age	Clinic No.
	Dr. Buckley	Operator Dr.		Date and time of opt.	
Clinic	Dr. Smith	Checked by Dr.			
	Dr. Faulkner			Date of hemorrhage	
	Dr. White			a.m.	p.m.
	Upper				Date
Right	Middle	Clamp		Plug	1st hemorrhage
	Lower	Tie	Adenoid	Removal of	2nd hemorrhage
	Method	Plug		piece	3rd hemorrhage
	Upper	Suture			4th hemorrhage
Left	Middle	Removal of			5th hemorrhage
	Lower	piece		Good	
			Patient's condition	Fair	1st transfusion
				Poor	2nd transfusion

Chart 5.

Hoarseness following operation has been noted. Hematoma of palate after local operation. One peritonsillar abscess after local; one Vincent's angina; one diphtheria. On examination of 110 colored patients, it was found that 12 gave positive Wasserman reaction.

140 East 54th Street.

SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

II.—HOW TO OBVIATE FAILURES IN THE RESULTS OF PARANASAL SINUS SURGERY.*

DR. ROBERT E. BUCKLEY, New York.

I have considered the subject in two main divisions. First, a general consideration of sinus surgery is important, because for a great many years we have been under a strenuous criticism by the general practitioner because we do get failures in sinus surgery. I think we do, but I think he, nevertheless, has an exaggerated complex on the subject. It is possible to get good results in a certain percentage of cases of sinusitis on condition that we will pick and choose our cases carefully. The first and most important cause of failures is the ill choosing of the type of case we are to operate on. For example, we know today that practically every asthmatic shows changes in the nose, changes similar, if you will, to angioneurotic edema. Whether this is caused by the asthma or is in conjunction with something that goes along with the asthma is something we must determine before we operate on the asthmatic patient. Occasionally we do see a severe polypoid and purulent ethmoiditis that offers pretty good hope, but in general I believe far too many asthmatics are the victims of sinus surgery, with the resulting large number of failures. Likewise, until very recently I think we laryngologists ran rather wild in our judgment of retrobulbar neuritis. We know now that it is a pathetically small number of cases in which the sinuses are the cause of this disturbance, and such conditions as multiple sclerosis and intracranial complications are forging to the front. These are only some of the causes for failure in general, and only better judgment can correct them. When we do any sinus operation, regardless of what kind, we do mutilate that nose to a certain degree,

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and unless the result is going to more than compensate for that mutilation, we are not going to do any good, but we are actually hurting our patient.

To be more concrete and more concise, I have divided the operations into the various sinuses involved, and have taken up, first, the intranasal antrum operation. I consider this one of the best operations we use, if it is done carefully and well. The intranasal antrum operation when well done offers in a fairly large percentage of cases good results, but that result cannot be expected by simply making an opening in the antrum. A permanent large opening must be made. The X-ray will show that in a large percentage of cases the floor of the antrum is on a lower level than the floor of the nose, so that this opening in the antrum must be flush, or as close to flush with the floor of the nose as possible. The first procedure in the intranasal antrum operation is a fracture and then a dislocation of the inferior turbinate away from the lateral wall, up toward and almost into the middle fossa of the nose. When this is done with a fairly large and wide speculum, a very good view of the nasoantral wall is obtainable. It is very easily possible to introduce various types of instruments into the antrum, such as Coakley curettes, and a large amount of the diseased tissue can be removed. In a large percentage of cases with this opening, Nature will take care of the rest. The inferior turbinate will frequently be seen to practically cover up the opening that has been made in the antrum. If this is so, within a few short days, and certainly within a few short weeks, it will absolutely ruin the result. When this occurs, or it is seen that this will occur, a wedge section of the inferior turbinate should be taken out over the most dependent part. It will not give an atrophic rhinitis to sacrifice three-eighths of an inch of the inferior turbinate in all directions. I think this overhanging inferior turbinate itself ruins more intranasal antrum operations than any other single factor.

Hemorrhage is a cause of failure, too; at least, of temporary failure. Here I think two things must be kept in mind. The posterior third of the inferior turbinate has a very large blood supply. Any damage to it is bound to produce a great deal of bleeding. At the exact same level with the antrum in the floor of the sphenoid is the posterior palatine artery,

associated with the Vidian nerve. An instrument biting too far back may catch the posterior palatine artery, and produce rather severe bleeding.

I am going to go rapidly to the Caldwell-Luc operation. Some of the men here know that at times I think it is done too often. There is an anesthesia following the Caldwell-Luc operation that sometimes lasts for five or six months, and the patients resent it a great deal. It is now an established fact that the mucous membrane lining the antrum is practically replaced as it originally was with a reformation of the ciliated epithelium, so it is no longer necessary to attempt to curette the antrum when doing a Caldwell-Luc operation. The membrane and polypoid tissue should be removed *en masse*. Next, in taking down the antral wall, if taken down too far it may catch the branch of the superior dental artery, and also the nerve. This latter causes the anesthesia of the teeth which lasts so for many months, and the former causes a hemorrhage which can be controlled only by bone wax. By going too far back we take a chance of catching the sphenopalatine artery. Having gotten into the antrum and taken out the lining membrane, a bony section of the nasoantral wall is taken out, but too frequently the mucous membrane is left in. If this membrane is left in it acts as a dam, and ruins the result. Here again, I think the inferior turbinate must be taken out, or rather a portion of it, at the most dependent part of the window. The Caldwell-Luc operation, in general, should never give a closed window. It should be a large and permanent window opening.

To go on to the ethmoid operation, it is difficult to speak of it without being paradoxical, because one done partially gives a very unsatisfactory result, but we know we cannot do a complete intranasal ethmoid operation without taking too great risks. Incompleteness, therefore, is the first cause of failure. I do not know how to get around it. I think the best way is to do a partial ethmoid first, then go into the sphenoid, get the landmarks, and then complete the ethmoid. One cause of failure is meningitis. Here the cause of failure is often the same; namely, working on the wrong level in trying to locate the sphenoid. The tendency, particularly with the patient lying down, is to look for the sphenoid too high. The second cause of failure is hemorrhage. Hemorrhage here

in a large percentage of cases is produced by taking down the anterior wall of the sphenoid laterally, rather than toward the midline. The walls can be taken down in absolute safety close to the septum. Laterally in the floor is the sphenopalatine artery. The other hemorrhage which causes annoyance is the anterior ethmoid artery, going across through the anterior ethmoid foramen, and passing upward through the cribriform plate, and down on the other side to the crista galli. There may be a perforation of the orbital plate, and corresponding puncture of the anterior ethmoidal artery behind the orbital capsule, causing hemorrhage behind the eyeball, with resulting blindness. The procedure if this happens is to strip the orbital capsule, and control the hemorrhage.

We should do nothing near the brain plate if we cannot actually see what we are doing. It must be done under direct vision. Another point to obviate failure, or possible complications, is to do a preliminary submucous resection, even if the septum is comparatively straight. A quick, high-up submucous resection will simplify the subsequent operation.

The external sinus operation, I think, has one particular cause of failure, a supraorbital ethmoid cell, particularly a deep one, well above the eye and running back almost to the optic nerve, which is so frequently interpreted as dura, or failure some place along there to pick up a large laterally misplaced cell. Incompleteness and lack of vision in the external sinus operation certainly mar the result. There should be a large incision made above or below the eyebrow, not through it, as that causes a wrinkled eyebrow, with the end of the incision extending down to the edge of the nasal bone. Next, seek a complete exposure, with no attempt to preserve the periosteum over the bridge. Complete separation of the orbital wall, and dislocation of the tear sac, and no attempt to preserve the tear groove, are not too radical. I have never seen a diplopia in the cases where the pulley has been completely detached. The orbital capsule can now be retracted, and the anterior ethmoidal artery identified. It is the only infallible landmark in this operation to show us where the limits of the brain are. Regardless of how it is done, or how well it is done, we can expect a possible recurrence after an external operation, and I do not think that recurrence is necessarily a failure.

829 Park Avenue.

SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

III.—HOW TO OBVIATE FAILURES IN THE RESULTS OF SIMPLE MASTOIDECTOMY.*

DR. MARVIN F. JONES, New York.

In answer to the implied question, "How to obviate failures in the simple mastoid surgery," the obvious reply is, "Do a thorough operation," "Know when to operate," and "Know enough to stop when the operation is finished." These statements are self-sufficient, although the second, *i.e.*, "Know when to operate," is probably the most difficult of performance. All physicians with sufficient training to be rated as specialists should have the ability to use good judgment in respect to all three.

DO A THOROUGH OPERATION.

If any one of you were asked, "Do you completely eradicate infected cells when you do a simple mastoid operation?" nearly all of you would confidently answer, "Yes." To those of you who seek enlightenment, I would suggest the following diversion: Do what you think is a complete mastoid on a cadaver. When you have satisfactorily accomplished your result, continue your excavation in the following locations: posterior and parallel to the facial canal as it runs from the horizontal semicircular canal to the stylomastoid foramen. Follow along the lower end of the sigmoid sinus; curette in the direction of the petrous apex, beginning in the angle formed by the sigmoid sinus posterior and the middle fossa above. Continue along the zygoma; explore posterior and parallel to the sigmoid sinus. Your self-confidence in your ability to completely remove all cells at every operation will receive a jolt, and you will be more hesitant in criticizing another operator's technique.

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My first rude awakening occurred during a simple mastoid operation which I was demonstrating to a group of matriculates at the Post-Graduate Hospital. The child was about 8 years of age. The child's history recorded a former mastoid operation at the age of 1 year. When I examined the cortex, I found only a small opening, which resembled the dehiscence frequently found in cases of subperiosteal abscess. I made a few pertinent remarks on what I supposed had been done at the former operation, *i.e.*, a Wilde's incision. The cells were well formed, and I concluded the mastoid had never been entered. After properly berating the former operator, the interne very graciously whispered that the first operation had been performed by myself. The only explanation I could offer to cover my chagrin was that the mastoid cells had continued development, and the cortex reformed after the original operation. This is not intended as an excuse to an operator who is not thorough, but to temper judgment on the original operator when a secondary mastoid becomes necessary.

KNOW WHEN TO OPERATE.

After reading an article in one of our leading journals, which decried doing a mastoid on a patient under 5 years of age, and after seeing cases which have been operated during the first few day of middle ear suppuration, knowing when to operate assumes major proportions in the subject of failures. Too early operative procedures frequently have a stormy course, with indifferent cosmetic results, while operations performed too late are apt to end unfortunately. We have seen the results postmortem on patients where the ears have been permitted to discharge over long periods of time without operative interference. It is the occasional case which does resolve spontaneously without sequela which leads us to hope against hope that our patient will do likewise. Failures in mastoiditis do not always result from the mastoid operation but for the need of it.

I would like to propose to parents an excellent insurance against fanaticism. Have it understood that in case of a fatal outcome, an autopsy will be done, the procedure to include an examination of the entire temporal bone, regardless of the supposed cause of death. The results of these examinations would limit undue and disastrous delay. The really difficult

problem today is how long can we delay, and how early should we perform the mastoid operation in order to reduce failures.

Another factor projects itself under the heading, "When to operate." This factor is the popular demand for all the consultants available. One good consultant with a background of age and experience in questionable cases is valuable. More than that number changes an operative problem into a medical discussion, with its typical "fiddling while Rome burns."

A FEW MISCELLANEOUS NOTES.

Preoperative and postoperative care have scarcely been stressed enough or their importance emphasized. In the preoperative care, we have a problem in which the general medical consultant or the pediatrician may be of great service. The eventual favorable result of a mastoid operation may depend upon the resistance of the patient. This phase should receive careful attention. The general postoperative care is as essential as the dressings. Too often, our gaping, nongranulating, sloughing wounds result from the lack of appreciation due to nutrition.

ERRATIC MEDICAL ARTICLES.

The publication of bizarre operations in standard medical journals leads to failures in surgery. Operators who fail to discriminate between practical advances in surgery and spectacular surgical theory are apt to have fatal results. I cannot blame the trusting reader as much as I do the magazine publishing the article. One way to eliminate this practice is feasible. Have all procedures presented before a reputable medical society before publication. At the time these operative procedures are presented, permit full discussions. In place of the dignified contempt of silence, make the criticisms frank and publish them with the article.

RECURRENCES NECESSITATING SECONDARY OPERATIONS.

We all recognize the fact that recurrent mastoiditis may come from infection in the mastoid which has not been eliminated at the first operation. Whether this is excusable or not lies largely in the conscience of the operator.

There is another field, however, which causes failure of permanent resolution. I firmly believe that pathology in the

upper respiratory tract causes many more recurrences of mastoid infection than infection which has been overlooked in the mastoid itself. Attention directed toward adenoids, sinuses and possibly the tonsils should materially decrease repeated mastoid operations.

I am sure you will excuse me for again mentioning allergy. It is a definite factor, and until we in our specialty learn to recognize its various manifestations, we will continue to spray and operate without relief. When we have learned that there is a local pathology which can be corrected by readjustment of a disordered systemic chemistry, we will be spared many of the distressing recurring failures resulting from operation.

I mentioned in my first paragraph as a third method of obviating failures: Know when you have gone far enough, or when to stop. I will now apply it practically.

121 E. 60th Street.

Professor Portmann, of the University of Bordeaux, France, will conduct an intensive course in Oto-Rhino-Laryngology in Washington, D. C., from Jan. 31 to Feb. 6, 1935, both dates inclusive. Dr. James A. Flynn, 1511 Rhode Island avenue, N.W., Washington, D. C., is in charge of arrangements. Direct all inquiries to him.

SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

IV.—HOW TO OBVIATE FAILURES IN THE RESULTS OF RADICAL MASTOIDECTOMY.*

DR. JOHN RANDOLPH PAGE, New York.

A safeguard against failure with the radical mastoid operation is to keep clearly in mind the things that go to make a satisfactory result. These things can be divided into those that have to do with the tympanic part and those that have to do with the mastoid part of the cavity.

The tympanic part should be wide and flat, with a closed Eustachian tube and thinly dermatized inner wall.

The mastoid cavity should be small and slope towards the meatus. There should be a low facial ridge between it and the tympanic part, and there should be no pocket in the tip.

The meatus should be large and inconspicuous.

Such a cavity is often self-cleaning and may go for years without attention. It is comparatively easy to get this result in cases that have a naturally wide tympanum.

In a case that has a narrow deep tympanum one has to widen the tympanum as far as possible on all sides and flatten the ridge, the floor and any bulge there may be to the anterior canal wall. When this is done, the removal of the anterior lip of the Eustachian tube gives additional width to the cavity and room to see to work well into and around the tube.

Different methods may be used in different cases, but no method is apt to succeed, without prolonged after-treatment, unless there is room gained to work in safely. It is well to bear in mind that as serious harm may be done with the back of a curette in this region as with its front.

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With a wide flat cavity, the necessary work in and around the tube can be safely attended to with whatever instruments one finds most effective. For the inside of the tube throughout its length, Yankauer Eustachian curettes are safe and satisfactory. I have never attempted to block the tube with knotted catgut pulled into it through the pharynx, nor have I ever cut a pedicled skin flap to cover it. Dr. Joseph Kelley's suggestion to place over the tympanic orifice a rather thick, narrow piece of skin, cut from the margin of the mastoid incision, is well worth trying. It has the advantage of being a prompt and simple way to close the tube and gain a skin graft when it takes, and of doing no harm when it fails to take. Except for this, I have not used a primary skin graft in many years, although I use skin grafts for the tympanic part of the cavity a week or more after the operation in many cases.

Regarding the mastoid region, a small cavity that slopes towards the meatus and has no pocket behind the facial ridge may be obtained even in large mastoids by removing the tip almost to the exit of the facial nerve at the stylomastoid foramen, and the bone upward and backward along the digastric muscle. This will allow the soft parts and neck muscles to fill in behind the facial ridge, where otherwise a pocket might be formed. The upper posterior part of the cavity can be further diminished in size by beveling its bony margin above and behind until it is well flattened so that when the posterior wound is closed the tissues fall close to the inner plate. It is better to let this part of the cavity take a long time to heal and have a small self-cleaning cavity, than it is to have a large cavity completely covered with skin in a short time, that requires attention every few weeks forever afterwards. For the same reason, it is better to confine skin grafting to the tympanic part of the cavity and allow every opportunity for the mastoid portion to contract and diminish in size before it becomes dermatized. This is a fact not generally appreciated, and the mistake is made of skin grafting a large cavity which prevents its diminishing in size and becoming a self-cleaning cavity.

"In my hands," as Dr. Gruening used to say, a satisfactory and inconspicuous meatus of uniform size can be cut in an adult or a child with this instrument, which modesty almost

prevents my recommending, since I myself designed it and seem to be the only one so far to appreciate its value. It is embarrassing to state that, although it was presented to the surgeons at the Manhattan several years ago, not even the members of my own clinic find occasion to use it. I find it very useful. There is no bleeding when it is used, so one can see well to further shape the flap when that become necessary. With it there is also no danger to an exposed sinus.

Most of us realize that while life may be saved by simply cleaning out the mastoid and tympanum, more than just that is expected of the specialist in this field. Special instruction and long practice on the cadaver is necessary. It is required, if not by law, by conscience, to qualify one to attempt this operation. When more realize this, there will be still fewer failures.

Keep in mind and work to get a wide tympanic cavity with a closed tube, a low facial ridge and a small mastoid cavity that slopes towards a large oval meatus, and so avoid failure.

127 East 62nd Street.

SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

V.—HOW TO OBVIATE FAILURES IN THE RESULTS OF BLOOD VESSEL SURGERY.*

DR. DUNCAN MACPHERSON, New York.

When our Chairman assigned to me the subject of blood vessel surgery, it probably was not his conception that I should discuss the constitutional diseases accountable for failures. So I shall take for granted that the patient is at least a fair surgical risk and that the surgeon knows his anatomy and fundamentals as well as the average — also his surgical guides, linear, muscular, bony, nervous, vascular sheath, pulsation, color of the vessels, etc.; also, that he will in particular cases fortify himself by anticipating dangers and preparing with instruments or other armamentarium that might be needed even if not likely to be used.

Poor operating room assistants are a menace to every operation where they assist — the proper instruments are not available, ligatures are not suitable, solutions are not at hand, the temperature of the operating room varies, lights are out of order, electrical appliances need last minute attention, etc. Poor anesthetists who have no proper training in physiology, anatomy and diagnosis are incapable of giving aid when emergencies arise and contribute their quota to failures.

Some of the related errors included in this paper I have committed either in civilian or war practice, and therefore speak with feeling about them.

Bruising of tissues is to be avoided, as is bruising of blood vessels. As between a choice of time saving and bruising, we can usually sacrifice time. Rough handling and tearing of connective tissue lessens blood supply and tends to pro-

*Read as part of a Symposium before the New York Academy of Medicine, Section on Otolaryngology, Jan. 17, 1934.

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mote infection. Bruising the outer coat of a blood vessel, from which the middle and inner coats derive their nourishment, weakens the circular coat and may lead to aneurysm. Surgical incisions should be sufficiently roomy. Many accidents result from insufficient operative space.

Veins and arteries are distinguished from each other by their relative blue and pink colors. The vein bulges, if not thrombosed, on being pressed — distal to the centre of circulation, while an artery if bulging at all will expand proximal to the heart if pressed upon. Beware of anomalies; for instance, the internal carotid in the neck may be closer to the trachea than the external, although normally it is further out from the median line. Be prepared to prove the identity of the structure being dealt with. Do not tie a carotid, mistaking it for a jugular — which has been done, but is not one of my failures referred to. Bear in mind that septic temperatures may come from small venous channels, and that all cases of mastoiditis carrying a septic temperature may not prove to be a lateral sinus thrombosis or require ligature of the jugular, although in such cases of mastoiditis the sinus should be exposed. This audience probably knows that pulsation of the sinus does not eliminate the possibility of a clot. Ligature and excision of the jugular where the sinus alone is involved is still controversial territory and I shall not discuss it. Anomalies of the carotids and their branches bearing on adenoid and tonsil operations should be in the minds of operators, and preparations made for such complications as they might be responsible for when present.

The following are necessary habits to follow: Proper material for ligatures. Use heavy silk for tying large arteries — not catgut. Catgut is slippery, is stiffer and more apt to unravel its knot, and may absorb before the blood clot becomes fixed and organized. The silk is fixed and permanent about the vessel, doing less harm as a foreign body than the above failures of catgut.

2. Tie at least one-half inch away from any sloughing area in the wall of a vessel. The general surgeons felt in France that if they were able to tie a femoral, for instance, one-half inch away from the macroscopically diseased area, they were safe from later hemorrhage due to extension of the slough.

In one case in which I first tied the lingual for bleeding at the base of the tongue and later the external carotid and the internal jugular because of spreading infection into the neck, and in which case I wanted to block off the lateral sinus, inasmuch as the tie of the jugular was necessarily very high but about one-half inch above the infected area of the venous wall, the consultant, a general surgeon of large war experience, from Cairo, said they felt safe if they got a ligature one-half inch away from the diseased area and advised against obliterating the lateral sinus. The man died of the most profuse hemorrhage that I saw during the war, about 30 hours after the operation. The consultant then said he thought the hemorrhage was due to faulty tying of the carotid, suggesting the following mistakes that I might have made: tying too near the crotch or too near a branch, using too thin a ligature, and tying too rapidly. The thin ligature or the rapid tightening down on the ligature would cause snapping of the internal and middle coats and permit the blood stream to pound against the external, resulting in an aneurysm, rupture and death. He cited the following experience that had happened to one of his other men, who tied the external carotid. Oozing followed, and he tied the common carotid. Oozing again ensuing, he tied the subclavian, and as bleeding followed that, the consultant said, "I take off my hat to anyone who ties the innominate," but he did that. At the autopsy it was found that the form of aneurysm described above was present at every tie, but I would note that at each tie the flow was seepage, giving the surgeon plenty of time to tie further down, while in my case the gush was sudden with death immediate, suggesting venous break. The experience occurred during the March, 1918, German Push and I was too busy to get or do an autopsy, much to the discomfiture of the consultant. I had similar experiences in two other cases, and from the three I would, under similar circumstances, deduce the advisability of obliterating venous branches and obliterating the lateral sinus as well.

In tying, identify and isolate the pneumogastric nerve. In tying the external carotid, identify it by placing a ligature under it, lifting it and noting the obliterating of the facial or temporal pulse. Tie a half inch away from the bifurcation and from the superior thyroid artery. The slowing of the pulse, from 130 to 60, noted by some on exposing the bifur-

cation I have not noted — and doubt that it is of much general practical significance.

In handling large vessels, tying with the thumb and finger is, I think, the best method to pursue, inasmuch as you get the sensation of force produced and also can feel the effect of the ligature if it snaps the intima.

In older people with hard arteries some surgeons place the ligature on large arteries at the distal end, and another not very tight, one-half inch or so nearer the heart to break the force of impact of blood against the first.

In tying the internal carotid, the vessel, according to Eagleton, should be closed off gradually, first lifting it, noting the effect on the cerebral circulation, and, if I understand him aright, paralysis and severe cerebral anemia noted in tying common and internal carotids will be obviated to a degree, as the danger is due to closing off the blood before collateral circulation gets a chance to exert itself. In fact, some surgeons feel that in ligaturing of all large vessels, pressure of some sort should be practiced for a sufficient length of time to permit of establishing collateral circulation.

Some years ago, a patient was admitted to the Aural Service of the Post-Graduate Hospital with bleeding from the ear. The house surgeon reported it to the attending and volunteered the information that the patient also had a peritonsillar abscess. The attending told him to open it, which he did and about a quart of blood spurted out. The patient had had a peritonsillar abscess for several weeks previous to admission, which ulcerated through into the neck. I prepared to tie the carotid but another hemorrhage supervened and he died before we could do it. An autopsy was not permitted, but I believe had I tied the carotid I would have been in error, the vein being more likely to have been affected — as it is thinner and more vulnerable. Besides, the bleeding from the ear I believe came from an ulcerating bulb.

Tying of the external carotid in nasal hemorrhage I have seen to be successful in arresting the hemorrhage and also have seen it make no impression. There is free anastomosis between both external carotid branches in the nose. Nevertheless, it is a practice to be resorted to in stubborn cases. Bear in mind that hemorrhage may be temporarily arrested

but with the establishment of collateral circulation it may recur. However, it may give sufficient time for the building of a vascular clot. Hemorrhages from the nose in postoperative cases are, I think, often due to the lip of a small blood vessel, temporarily clotted, slipping the clot and being caught on a spicule of bone or a septum of a sinus, thereby preventing its retraction. In such cases the punch or curette may be of value, if packing fails, to release the blood vessel.

When best to operate in acute cellulitis to avoid blood stream infection cannot be put in articulate language that will cover more than a few cases. Any given case may require to be dealt with by practicing the art of medicine, while another may give the cue only by physical and laboratory findings. Experience in handling of such cases is valuable, as in all serious phases of disease.

Ligature should be placed above and below the section of veins and arteries. The establishment of collateral circulation resulting in later hemorrhage should always be borne in mind.

All bleeding should be stopped before opening a larynx or trachea — except in emergency cases.

Tension on larger vessels in the long axis detaches the circular coat, establishing a weak spot that may result in aneurysm.

Neck infections with jugular thrombosis following tonsillitis, pharyngitis and scarlet fever are not uncommon and evidence of blood stream infection is to be looked for in any neck infection accompanied by septic temperature and may require blood vessel surgery.

In hemorrhage from the inferior dental artery or from any artery following a bony canal, catgut forced into the canal was recommended by Bryant.

In operating on nasopharyngeal fibromata or on large vascular tumors of that region or of the upper jaw, one should have the external carotid under control. Placing a ligature under the vessel is advisable either as a method of lessening the flow of blood through it or preparatory to tying should the condition demand it during the operation.

Some years ago, in removing a fibrosarcoma involving the upper jaw and the nasopharynx, hemorrhage was excessive. No information was forthcoming from the anesthetist — the tissues suddenly blanched and the patient became pulseless. Foreseeing the likelihood of hemorrhage, we had prepared for a venous saline and but for the rapid action of a particularly quick thinking and quick fingered senior house doctor, F. W. Hayes, the patient would have died instead of living, now about eight years after the removal of the sarcoma. This one case was enough to cause me to have the carotid under control in any case where before the beginning of the removal of a growth excessive hemorrhage would be anticipated, or if during the operation hemorrhage became excessive.

It is my feeling that in any acute cellulitis, danger of systemic absorption and local extension is increased by too early opening before walling off takes place. In peritonsillar abscess I have seen chills and high fever follow attempts at too early evacuation of pus. Forcible opening of scissors and forceps traumatizes the tissues, causes increased local swelling, internally and externally, with aggravated systemic symptoms.

However, in mastoiditis, for instance, it is sometimes a nice balance of judgment that decides accurately the best time for operative interference, taking into consideration the degree of Nature's walling off process as well as the general condition of the patient, balancing all, including the possibility of sinus disease supervening in case of delay.

In unexpected hemorrhage, finger pressure against bony structures may be invaluable. It is surprising, too, what violent hemorrhages may be successfully dealt with temporarily by gauze pressure in certain localities.

Cavernous sinus infection results from pharyngitis and mastoiditis; travels also from the eyelid or ethmoidal cells, also from the teeth or facial and angular veins. No cutting should be carried out in furuncles of lip or nasal vestibule. Use of small poultices and raisins in the vestibule will hurry local breaking down and may prevent involvement of the cavernous sinus.

It should be borne in mind that edema of the lids and exophthalmos may not be present until there is complete oblit-

eration of the sinus. A floating, suspended clot may cause symptoms of infection and later pyemia, but the cases seen by me did not have marked rise and fall of temperature, symptomatic of and so common in lateral sinus thrombosis. This may also be true of longitudinal sinus thrombosis where early extension to the meninges takes place.* Differential diagnosis is frequently difficult even with so-called classical symptoms where it must be differentiated from orbital abscess and cellulitis. A history of a furuncle or wound on the lip, nose or cheek will give one a lead more suggestive of cavernous sinus thrombosis than would a history of ethmoiditis, pharyngitis, antrum, teeth or neck infections.

In France, with the Canadians and a service that gave me about 200 head operations, with a fair proportion of necks, varying in point of time after injury from an hour to indefinite periods up to a week, I did not learn much about arterial brain hemorrhages. Longitudinal sinus wounds, torcular and lateral sinus wounds were considered and handled on their individual requirements and as complications of even more severe injuries. This also held true of arterial hemorrhage.

Aneurysms of the carotid, I tied. Paralysis or acute cerebral anemia from tying of the common carotid, I saw in at least one case in my practice. I have resorted to it in other cases.

114 East 54th Street.

*Eagleton: Cavernous Sinus Thrombosis. 1926.

SYMPOSIUM: HOW TO OBVIATE FAILURES IN RESULTS OF SURGERY IN OTOLARYNGOLOGY.

VI.—HOW TO OBVIATE FAILURES IN THE RESULTS OF SURGERY OF BRAIN ABSCESS.*

DR. LEO M. DAVIDOFF, New York.

It is apparent that the best way to obviate failures in their treatment is to prevent the formation of brain abscesses, but I do not feel capable of instructing this audience in such prophylaxis. From what we have already heard, the members of this Section are very well aware of the dangers and the possibilities of such abscess formation.

Assuming that a certain number of abscesses of the brain are unavoidable, how often does the surgical treatment of this condition fail? The answer to this question is appalling. At least 50 to 75 per cent of all brain abscesses that are treated surgically, die. Almost 100 per cent of those that are not treated surgically also die.

In order to arrive at some answer to this serious problem, I have analyzed the records of 40 cases of brain abscess that were admitted and treated at the Neurological Institute between 1925 and 1933 on the service of Dr. Charles A. Elsberg. Of these 40 cases, nine were admitted to the hospital and died before operation was undertaken; 31 were operated. Of the operated cases, 13, or 41 per cent, recovered, and 18, or 59 per cent, died. In looking over the nine cases that died before operation was undertaken, I found that six of these were patients who presented themselves in a moribund state on arrival at the hospital. All of the six showed signs of extension of the suppurative process from the abscess of the brain into the subarachnoid tissues or into the ventricles. Three of these were autopsied and, as anticipated, were found to have pus extending into the ventricles, as well as into the

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subarachnoid space. The other three were not examined postmortem, and the extent of the pathological process was not determined. The remaining three were individuals in whom the diagnosis of brain abscess was not certain. There was some question as to whether they were suffering from brain abscess or tumor, and the disease was not localized. All of these died before localization was made and at postmortem proved to have brain abscesses. I shall mention in a moment something further concerning the failure of localizing the abscess.

Let me go now to the 18 cases of brain abscess that were treated surgically and failed to recover. I thought at first that some connection might exist between the organism that caused the abscess and the chances of the patient to survive. There had been bacteriological studies in 23 cases; eight were recovered cases, and 15 were cases which ended fatally. I found that most of the common pyogenic organisms were represented in the 23 abscesses. Staphylococcus aureus was found in six cases, of which five died and one recovered; streptococcus hemolyticus in five cases, of which two died and three recovered. There were two cases of pneumococcus infection; both died. The interesting thing is that in four of the cases studied bacteriologically, no organisms were recovered from the pus submitted to the laboratory. One would expect that all of these cases would recover, but two of them died and only two recovered.

In examining further the cases that died following the operation, I found three in which the localization was not made accurately. These were cases in which a diagnosis of brain abscess was made, the approximate localization was also made, and an exploration failed to strike the pus. The patients eventually died, and at postmortem the abscess was found to be within the vicinity which had been explored, but the exploring needle had apparently passed by the abscess. The failure in these three cases, I think, is somewhat different from the failure in those cases in which operation was not done because localization was not made. In the cases where failure to strike the abscess occurred in spite of puncturing in the neighborhood of the abscess, I believe the operator in each case failed to recognize the pathway of the extension of pus from the otolaryngological focus. I think if we keep in

mind the pathway which purulent material takes from an ear, for example, to the temporal lobe, we are much less likely to fail to strike the abscess. In regard to the cases which died before localization was made, at the present time with the improved method of encephalography, I think it is safe and indicated in such cases to use this method to localize the disease and attempt to cure it by surgery.

Of the 18 cases that died after operation, two further cases died as a result of meningitis, obviously spread at the time of operation into the subarachnoid tissues. I believe this can be avoided by carefully packing off the subarachnoid space before the abscess is punctured. Two more cases did well for a short time after operation and then died. Postmortem examinations showed multiple abscesses. This fortunately is not a very frequent occurrence in abscesses that extend from local foci. Both of the cases here recorded were metastatic abscesses from foci elsewhere in the body. There now remain eight cases still unaccounted for. These I shall not take up in detail. Some of them were acute abscesses with a very short history, rapid progress of the symptoms, and very little or no capsule, in which the patient's chances were obviously less good than in the others. One or two of these were abscesses so deeply located in the cerebellum that one had to traverse a very thick portion of brain tissue before the abscess was reached. In general, however, I feel that in this entire group of eight cases, failure occurred probably because of inadequate evacuation of pus and inadequate sterilization of the wound.

I have been very much interested in brain abscesses for a number of years, and have combed the literature, I think, rather thoroughly. The reports, literally in hundreds, are extremely discouraging, but there is one book, published in 1893 in Edinburgh, which is a light in the desert. If this book were written by a lesser person than William Macewen, its value might have to be discounted. If it were a report on a lesser series of cases, even by Macewen, it could be largely discounted. He reported on a series of 25 cases, of which six were moribund on arrival at the hospital and died without operation. The other 19 cases were operated upon and 18 of them recovered in Macewen's hands! This is no accident. It is much too many cases, and certainly there is no

question of the veracity of the report. It, therefore, seems to me important to study in the greatest detail what Macewen did to get this astonishing percentage of recoveries. I think if one examines carefully the report of his technique, one will find, first, that Macewen knew the pathology of the spread of infection from the otolaryngological foci. He, therefore, entered the brain at the optimum point. Second, he never went through an infected focus. He always went through a clean area. Third, he always evacuated the abscess completely and repeatedly washed out the cavity until he considered that cavity sterilized. He did that with an antiseptic solution. In 1893, they were still using carbolic acid. I believe today we would use Dakin's solution. After he was convinced that the abscess cavity was empty and sterilized, he very seldom used a drain at all. This is a principle that has been recognized by many operators on brain abscess. It is the desire to evacuate completely the cavity that has given rise to the various speculae, Whiting's encephaloscope, King's method of marsupializing the abscess cavity, etc. The essential point is to empty the cavity, not only of free pus, but of necrotic material which contains bacteria. Once that is done, if the abscess cavity wall or capsule is so soft that it collapses, it was Macewen's practice to leave no drain at all. He very often describes, very naively, I believe, the manner in which he replaced the disc of bone removed by the trephine so that there would be no defect in the skull. He then applied the bandage and left it in place for three weeks, after which the bandage was removed, and a few weeks later the patient was sent home completely well. In the cases where the capsule was very old and thick and failed to collapse, he used a drain. In his time, they used decalcified chicken bone, which became absorbed. He also used rubber and glass. It apparently made no difference.

As far as my experience goes, last year I had the privilege of presenting to this Section of the Academy four consecutive cases of brain abscess, all of which recovered. They were all treated essentially by the method of Macewen. The cavity was entered, the pus completely cleaned out under inspection, the cavity carefully washed out with Dakin's solution until the fluid came back absolutely clean. In all these cases a drain was used, but very little drainage occurred. Had I more courage, I might have removed the drains sooner. I removed them in three weeks.

I believe the above points are the secret of Macewen's success in the treatment of brain abscess. I realize that his method cannot be applied to all cases. There are deep-seated abscesses where a good look at the cavity is impossible. There are cases in which the abscess is so tough it moves away from your instrument. But I think that wherever possible, if this principle is applied, recovery from brain abscess is much more likely than was true heretofore.

Neurological Institute.

THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The Forty-first Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., will be held in Toronto, Canada, on June 3, 4 and 5, 1935, during the week following the meetings of the American Otological Society, the American Laryngological Association and the American Bronchoscopic Society, in Toronto. The meeting of the American Medical Association will be held in Atlantic City, N. J., during the week following our meeting.

The scientific program is already being prepared and those Fellows who are planning to present papers should so notify the Secretary promptly. Twelve papers will be selected for presentation.

Copies of the *Transactions* for the past five years are available to those Fellows who have not already received them, without cost. Application for them should be made to the Editor of the *Transactions*, Dr. Lyman G. Richards, 319 Longwood avenue, Boston.

Will you please note the change of address of the Secretary, Dr. Robert L. Loughran, Sharon, Conn.

THE PART PLAYED BY THE TONSILS AND ADENOIDS IN THE ETIOLOGY OF RHEUMATIC FEVER.*

DR. GEORGE M. COATES and DR. WILLIAM GORDON,
Philadelphia.

The treatment of this subject, assigned to us by your Committee on Program, may be divided into a discussion of two main points, as follows: Does infection in the tonsils (and adenoid) cause rheumatic fever, or other rheumatoid diseases; and how is infection of these organs to be recognized? Our title limits us to this consideration, but the fact must not be overlooked that, after all, infection of the teeth, nasal accessory sinuses, even the ears possibly, as well as in other more remote parts of the body, may be either the principal or contributing factor. If we bear this in mind it will explain why removal of tonsils may fail to give relief even if they happen to be one of the primary factors.

As for the first question, does tonsil infection cause rheumatic fever, opinions either pro or con can be collected in numbers to support any individual viewpoint, but statements and investigations made by men standing high up in their specialties are worthy of consideration and we shall introduce here the opinions of a few whom you will all know and to whom you can give credence. Before doing so, however, may we very briefly review a few of the most important features of the clinical anatomy, as well as histopathology and clinical pathology of the faucial tonsils and adenoid; for these two structures must be considered as a clinical entity.

The faucial tonsils, two in number, consist, in the main, of a fibrous capsule from which trabeculae pass into the parenchyma of the tonsil. These trabeculae carry blood vessels, nerves and efferent lymphatics, *i.e.*, lymphatic channels away from the tonsil, there being *no afferent lymphatics*. The parenchyma of the tonsil consists of the germinating follicles and

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the interfollicular tissue, all of which is located between the trabeculae. The free surface of the tonsil presents ingrowths of surface epithelium of the stratified squamous type. This epithelium dips in to form the crypts. Lymphoid cells infiltrate the cryptal epithelium to such an extent that there may be, and usually are, two or three layers of epithelium separating the lumen of the crypt from the tonsil parenchyma.

Grossly, the faucial tonsils are two more or less almond-shaped masses of adenoid tissue placed one on each side of the oropharynx, and between the pillars of the fauces. In childhood, they are more globular but recede as age advances. On the free, or mesial surface, there are present a number of pits or crypts varying in depth from 1 mm. to 1 cm. or even more, the larger crypts often expanding below the orifice so that retention of large quantities of secretion or debris can take place. Not infrequently, the mouths of these crypts become sealed by reason of acute or chronic inflammation, and the sac then has no outlet and may take on some of the characteristics of an acute or chronic abscess.

The pharyngeal tonsil, or adenoid, often spoken of as the third or Luschka's tonsil, occupies the vault of the pharynx, at times almost completely filling this cavity. With the growth of the individual, the tendency of the adenoid to atrophy is more marked than in the case of the faucial tonsil and, indeed, it is comparatively rare to find any great amount remaining, after adolescence. The adenoid is a lobulated organ, having a succession of grooves and ridges, ranging in number from three to five. While focal infection is often attributed to the faucial tonsils, the adenoid as a focus is commonly overlooked, although it may be the true source of the disease. This is because the adenoid is out of sight behind the velum of the palate and because, in adults, it is assumed that it has undergone atrophy.

Simple inspection of the fauces will usually fail to determine with any accuracy whether or not a tonsil is infected, and it may be stated here that an absolutely negative report is never possible until the tonsil has been removed and studied histologically and bacteriologically. Small sealed crypts deep in the tonsil substance are not uncommon and are often impossible of detection, even with the transillumination method of French. A careful inspection of the fauces will often reveal a purplish red tinge limited to the anterior pillar,

which is supposed to have some pathological significance, especially where the infecting organism is the streptococcus. Often masses of cheesy debris may be observed filling the crypts if the anterior pillar is withdrawn by a pillar retractor. If, in addition, pressure is made external to the meridian of the tonsil, cheesy masses or liquid, grayish or yellowish, pus or mucopus, will flow from one or more crypts, the superior ones in the upper pole being those in which this secretion is most apt to accumulate. Histological examination presents a variety of cellular elements, notably plasma cells, eosinophiles and polynuclear leukocytes in the tonsillar parenchyma and in the crypts. Adenoid examination is much less frequently done, but where undertaken, the presence of large numbers of polynuclear leukocytes is reported by the pathologist. Numerous strains of bacteria are almost always observed and putrefactive changes take place in the crypts, which are themselves excellent incubators for pathogenic bacteria. Allen J. Smith has told us that here micro-organisms usually multiply and at the same time elaborate toxins. This results in local lesions being produced in the crypts and in the parenchyma. As long as the body resistance and the immunologic powers of the individual are able to cope with the bacteria and their toxins, no deleterious systemic lesions are produced. As soon as these vital powers are diminished, however, the bacteria and their toxins are enabled to attack important organs and set up irreparable pathologic changes. The toxins may enter the parenchyma from the crypts and either be absorbed by the venous capillaries, or be removed by the efferent lymphatics. Soluble toxins may be absorbed directly from the tonsil into the blood stream, thus accounting for the early intoxication in acute tonsillitis.

It is not generally appreciated that the adenoid may have just as serious focal possibilities as the tonsils, teeth or sinuses. Bacterial multiplication readily takes place in the deep crypts, in which, after removal, pus is often noted. From this source infection may travel down the nasopharynx to the oro- and laryngopharynx and to the tracheobronchial tree. Cultures taken from the crypts of removed adenoid masses show growth in 100 per cent of cases.

The question now arises: How can we tell when a given tonsil is infected? In many instances this is a difficult diagnosis to make. It may be perfectly easy to say, at a glance,

that this particular tonsil is infected — the objective signs are conclusive. But can we *ever* say that a given tonsil is *not* infected? We do not think so. While operating upon tonsils and adenoids, sometimes on suspicion only, the laryngologist is not infrequently surprised to find infinitely more evidence of infection in the depths of the crypts than the clinical examination revealed. This is due to the fact that the snare or guillotine squeezes the tonsil much more firmly during removal than can be done by methods of examination. Sectioning of the tonsil after removal will also often reveal unsuspected purulent collections.

The Le Meé questionnaire, referred to in more detail later on, asks, "On what signs do you base your opinion that the tonsils may be a cause of secondary infection?" and the collective answer is as follows:

1. Escape of pus from the crypts spontaneously, or by pressure on the anterior pillar, or by aspiration (50 per cent of the replies). It should be noted that the majority of the respondents assign little value to caseous masses in the crypts, considering this significant only when surrounded by purulent secretions.

2. Repeated exacerbations of acute tonsillitis (Hajek's dictum) or peritonsillar abscess (65 per cent).

3. Congestion of the anterior pillars (50 per cent).

4. Cervical adenopathy (40 per cent).

5. Presence or retention of caseous matter (20 per cent). In the opinion of those who gave this answer, the caseum unaccompanied by pus suffices to characterize the tonsil as a "bad" or "responsible" one.

6. Buried, small or "adherent" tonsils (20 per cent). (George B. Wood protests against the term adherent. What is meant is that by the deposit of fibrous tissue around the capsule, due to repeated acute, or chronic, infection the tonsil is more or less immobilized, and dislocated from its fossa with greater difficulty.) The buried, small or adherent tonsil is particularly suspected by English and American writers.

7. Simple tonsillar hypertrophy with enlargement of the cryptal orifices and eventually the presence of small ulcerocongestive lesions at their borders (10 per cent).

8. Bacterial examination (5 per cent), especially if the streptococcus hemolyticus is recovered. Judson Daland has often spoken and written advocating this method of diagnosis. His dependence is placed upon the type of organism recovered and the number of the colonies produced.

Years ago, John Nolan Mackenzie, of Baltimore, startled the profession with his paper entitled, "Massacre of the Tonsils," and Shambaugh has recently again protested against indiscriminate tonsil removal. The latter believes this is justified only when actual pus can be demonstrated, for then they may constitute a menace. He does *not* think that the presence of cheesy debris is an indication.

The writers feel that the pathogenicity of these cheesy concretions is at least a moot question, and that where present, the case should be studied with extra care.

To make a diagnosis of tonsillar infection then, we must make use of all the above mentioned points, but the clinical evidence should always be studied in its possible relationship to the history before the tonsil is condemned — that is, if we wish to be conservative, as, we believe, we should. It is easy enough, on the other hand, to make a diagnosis by agreeing with Beck, who stated years ago that all adult, and most adolescent, tonsils were infected, therefore a menace, and should be removed. It is, in our opinion, doubtful if a tonsil that has once undergone serious infection, ever undergoes complete resolution.

These we consider the high spots on which a diagnosis can be made: The history; the clinical evidence of infection in the crypts after careful search; cervical adenopathy, otherwise unaccountable; the bacteriology; the size and position of the tonsil; the condition of the remainder of the throat, particularly the anterior faucial pillars. Repeated examination may be necessary before a definite answer can be given.

That the tonsils and adenoid are very often infected no one will deny, or that they may act as a focus of infection for remote parts of the body in many instances. The question, for the purposes of this paper, now arises: What role do the tonsils and adenoid play in the etiology of rheumatic fever?

J. M. LeMeé, several years ago, sent out a questionnaire on this subject to clinicians all over the world. The referendum was not addressed to any particular group but to internists, pediatricians, orthopedic and general surgeons, ophthalmologists, otolaryngologists and pathologists. Nearly every medical centre in the world was represented. The replies of the respondents, several hundred in number, were carefully tabulated and printed in book form and are of interest as presenting a cross-section of world opinion. We give here a few of the most apropos questions and a summary of the answers to them.

Question: Do you believe that the tonsils may constitute a portal of entry and a focus for secondary infections?

Affirmative answers were almost unanimous.

Question: What place do you assign to the tonsil among other generally accepted foci; namely, the teeth, ears, sinuses, gall bladder and prostate?

Answer in four classes:

Class 1 (60 per cent of the replies) assigned first place to the tonsils. Class 2 (18 per cent) assigned second place to the tonsils, giving first place to the teeth. Class 3 (12 per cent) held that the tonsils shared equally with other foci. Class 4 (10 per cent) assigned first place to the tonsils in children and first place to the teeth in adults.

Question: Relation between the tonsils and various forms of rheumatism (acute, chronic and deformans)?

Answer: 1. Affirmative replies, 90 per cent. *a.* Almost unanimous for acute rheumatism. *b.* For a relation to chronic rheumatic infections, 45 per cent. Improvement here is very frequent after tonsillectomy. *c.* Rheumatism (arthritis) deformans, 5 per cent. Tonsillectomy almost never followed by improvement.

2. Doubtful, 10 per cent.

Some of the details of this study have been purposefully gone into because of the painstaking manner in which it was carried out. The general conclusions arrived at by means of this referendum may be summarized as follows:

The tonsil appears to be the portal of entry for infection in most cases of acute rheumatism.

Tonsillectomy has an unquestioned influence upon rheumatism and rheumatic fever. In children, initial attacks are but half as frequent in those who have undergone tonsillectomy as in others of corresponding age and numbers. It cannot be promised that tonsillectomy done at an early age and before the occurrence of infection will surely prevent rheumatism, but it is considered one of the most effective therapeutic measures in cases of existing or active rheumatism.

Within the last year, Herbert Tilley,[3] the eminent British laryngologist who was our guest in Philadelphia not long since, spoke on this subject before the Fellowship of Medicine, and his conclusions were virtually the same as those given above for the LeMeé study.

Hill Hastings,[4] in 1921, gave a critical review of his results in cases where tonsillectomies were done upon adults for rheumatism. In an elaborate and painstaking study, he concluded that all adult tonsils, when present, are infected, and that the same is probably true of children. The necessity for a tonsillectomy depends, therefore, not solely upon the examination by a laryngologist, but upon a complete study of the patient to determine all possible factors that might be responsible for the invalidism. Routine tonsillectomy without such careful study of the cases is to be condemned and there is urgent need for group study of all tonsil patients and that a decision either for or against tonsillectomy is ill advised in any given case until this is done. He has often been disappointed in the results obtained in cases of tonsillectomy done for the cure of rheumatism.

Pemberton[5] has called attention to the fact that even though focal infection was responsible for many kinds of disease, many cases get well even *in the presence of surgical foci of infection.* This led to a series of detailed studies which were very revealing. Metabolic studies, glucose tolerance tests, blood chemistry, blood gases studies, studies of the blood vascular system, blood flow, sweat, body mechanics, and elimination were undertaken. It was found that each case had to be thoroughly studied and that no panacea existed for rheumatism. Many cases improved with proper dietetic measures, others after a tonsillectomy, still others from the use of hydrophysio-therapy. Some cases needed a tonsillectomy in combination with dietary measures, and others fractional doses of

alteratives to improve metabolism. The G. I. retention cases required improved eliminatory measures as approved by the gastroenterologists, while nonspecific protein therapy helped in a certain number. Brilliant results followed a restrictive diet considerably below what the body had been accustomed to, thus reducing the load and giving the body less to metabolize. His conclusion was that in addition to the removal of foci of infection, all measures calculated to normalize metabolism, promote blood flow, dilate the peripheral vessels, minimize toxemias, lessen the body load and promote elimination should be used in a properly studied and treated case.

While the tonsils may be proved to be, beyond question, infected, we are unable thus far to say with certainty whether or not they are the absolute, or primary, cause of the rheumatism in any given case. There may be multiple causes for the rheumatic fever, and they should all be dealt with as thoroughly as possible. The infected tonsil may cause or perpetuate another focus which may be directly responsible, as for example in the intestinal tract.

However that may be, whether the tonsils and adenoid are responsible in whole or in part for the rheumatoid syndrome, or whether they have no relationship to it whatsoever, once the fact that they are infected is definitely established, they should be removed. The writers wish to stress the importance of a blood study in each case, of a detailed survey by the internist, pediatrician, laryngologist and other colaborers in an endeavor to establish a well planned management.

In conclusion, from the laryngological standpoint alone, certain principles that were written elsewhere[6] may be emphasized here. Every part of the pharynx and fauces should be examined with care. Even after a tonsillectomy has been performed the fossae should be diligently examined for stumps, tags, regrowths, small crypts concealed behind the pillars or beneath scar tissue, compensatory hypertrophies of the lateral pharyngeal folds, isolated lymphoid masses on the posterior pharyngeal wall, infected adenoid remnants, infected lymphoid structures (the lymphoid apron or lingual tonsil) on the base of the tongue, whose importance has been emphasized by French, as well as for the gravitation of infected secretions from an infection of the posterior series of sinuses or an extension of inflamed mucosa.

It is obvious, therefore, that the laryngologist, by exercising meticulous care, may find a source of infection in the naso-, oro-, or laryngopharynx which should be judiciously removed, provided that opinion is shared by the internist, pediatrician or referring physician.

BIBLIOGRAPHY.

1. LeMee, J. M., and Worms, G.: *Les Foyers Amygdaliens*, pp. 1-15, 1931.

2. Shambaugh, George E.: Fads and Fancies in the Practice of Otolaryngology. *Trans. A. L. A.*, pp. 168-177, 1926.

3. Tilley, Herbert: Foreign Letters. *Jour. A. M. A.*, Vol. 102, No. 8, p. 629, Feb. 24, 1934.

4. Hastings, Hill: Tonsillectomies in Adults for Rheumatism with a Critical Review of the Results. *Trans. A. L. A.*, pp. 41-51, 1921.

5. Pemberton, Ralph: The Nature of the Influence of Focal Infection and the Means Necessary to Meet It. *Trans. A. L. A.*, pp. 141-161, 1923.

6. Coates, George M.: Focal Infection in Early Childhood as Seen by the Otolaryngologist. *Penna. Med. Jour.*, March, 1932.

1721 Pine Street.

PATHOLOGY OF CHRONIC SINUSITIS IN CHILDREN.*

DR. WILLIAM SPIELBERG, New York.

The histopathology of chronic sinusitis in children does not differ materially from that of the adult. In children, however, the involvement is less extensive and frequently confined only to one or two accessory cavities.

Clinically and pathologically we recognize two forms of chronic sinusitis, *viz.: 1.* Hypertrophic (hyperplastic, polypoid, etc.); *2.* Atrophic (fibrotic, sclerotic).

Although mixed forms of atrophy and hypertrophy exist, we feel after considerable observation of these atrophic changes in children, that from their very incipiency they are atrophic, and that as such they are to be differentiated from nasal atrophy that follows chronic hypertrophic rhinitis frequently seen in the adult patient.

In dealing, therefore, with atrophic rhinitis of the non-ozena type, we are concerned with a distinct and separate pathological entity differing from an atrophic rhinitis associated with or following the chronic hypertrophic lesion.

When examining a patient rhinoscopically, it is not at all difficult to tell from gross appearances of the nasal tissues what the predominating lesion is. It either tends toward hypertrophy or atrophy. The nasal mucosa and its turbinates being a mirror picture of the sinus mucosa, reflects therefore the pathologic state of the latter. Both types of sinusitis may be associated with a superimposed suppuration. Vasomotor and allergic rhinitis will not be included in the present discussion. We have never seen an allergic rhinitis with an atrophy of the nasal mucous membrane.

1. Hypertrophic sinusitis: a. Gross pathology. The gross pathologic picture is that of a general hypertrophy of the nasal mucosa accompanied by congestion and turgescence and

*From the Otolaryngological Department of Beth Israel and Gouverneur Hospitals.

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loss of tone. The inferior turbinate bodies are mostly involved and are found greatly enlarged, impinging on the nasal septum, resulting in complete nasal obstruction of one or both nares. When superimposed by suppuration, the nasal chambers are filled with mucopus or pus bathing the turbinates. On shrinking the nasal mucosa with adrenalin solution (1-1000), the middle turbinates come into view. These are found congested and hypertrophic, but to lesser degree than the inferior turbinates. In the presence of an ethmoid infection, the anterior tips of the middles are covered with pus or

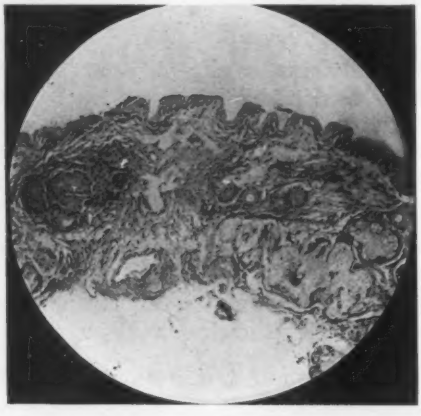

Fig. 1. Low power antral mucosa. Chronic antral suppuration of long standing in a child of 6 years. Note, total absence of epithelial lining; marked thickening of subepithelial coat (hyaline layer); fibrosis of stroma causing pressure and crowding of glands; glandular atrophy.

a dried glistening film of secretion resembling a crust on the removal of which the mucosa frequently present polypoid changes. Secretions can frequently be seen in these cases coming from the region between the ethmoid and middle turbinates, *i.e.*, upper middle meatus from hiatus semilunar, nasofrontal duct or hiatus antra. The pus filling the nose can be seen dripping posteriorly into the epipharynx and into the throat and larynx. Examination of the throat reveals a marked postnasal drip of pus, a markedly irritated congested pharynx diffusely infiltrated with lymphoid nodules, particularly in the fossae tonsillaris, where a previous tonsillectomy has been

done, as well as large pieces of recurrent tonsil tissue. Laryngoscopy, when possible, frequently reveals dripping of pus into the larynx through the interarytenoid space or filling at first the pyriform sinus drips into the larynx by spilling over the arytenoids.

b. Microscopic pathology: Due to the length of this paper, brief reference only will be made to the microscopic pathology. The mucosa lining the nasal chambers and the accessory sinuses as well as its underlying bony framework has a definite architectural structure. This structure shows characteristic variations in accordance with its site or location. The pathologic changes, however, are much the same in the majority of cases differing somewhat in instances according to the degree of disturbed circulation, drainage and ventilation. For the sake of brevity, therefore, and due to this general similarity in the pathology, the latter will be discussed more from a general aspect and in accordance with the changes found in the various important layers comprising the nasosinal mucosa.

1. Epithelial layer. *2.* Subepithelial, hyaline layer or basement membrane. *3.* Tunica propria (stroma): *a.* Blood vessels; *b.* Glandular structure; *c.* Cellular elements. *4.* Periosteum. *5.* Bone.

The subdivision of chronic hypertrophic sinusitis and rhinitis into papillary, edematous, infiltrative, cystic, degenerative, polypoid, granular, etc., etc., is to my mind superfluous and unnecessary. One rarely sees a single specimen without being able to find almost any or all of the above forms; and if sufficient sections are made at various levels of several pieces of tissue removed from different parts of the nose and sinuses in the same individual, one is sure to find every type of lesion possible, of course, with certain exceptions, from the absolute normal to the most pathologic. It is also true that certain pathologic lesions will predominate, such as extensive polypoid degeneration of mucosa with marked cystic changes in them, marked edema of stroma, papillary hypertrophies, areas of intense round cell infiltration, extensive fibrosis and degenerative changes in the gland and epithelium structure, etc. But all these grades or forms are but the result of the same pathology, only in a progressed and altered state and still fall under the head of chronic hypertrophy. According to Manasse, a sharp differentiation of these forms is not

possible, because there are many mixed forms, indeed cases where in one and the same specimen all forms are united.

1. The epithelial layer: a. The epithelium of the nasal and turbinal mucosa shows moderate to marked hyperplasia. Where polypoid changes exist the epithelial layer may be hypertrophied to the extent of 10-12 layers of columnar epithelium piled up on one another with many infiltrating leukocytes making their way to the surface. Goblet cell formations is rather common at the surface, where they become

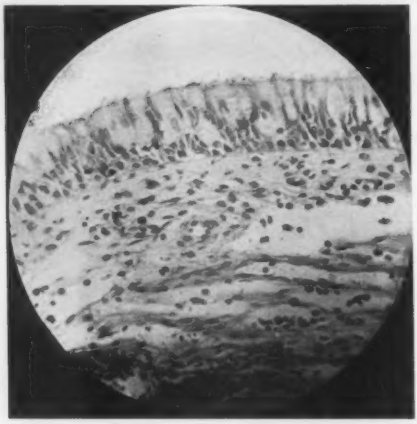

Fig. 2. High power ethmoid mucosa. Note, absence of epithelial cilia; marked dilatation of epithelial cells (goblet cell degeneration); marked edema of stroma with marked round cell and polymorphonuclear infiltration. Note that secretory activity is here carried on chiefly by the surface epithelium as there is complete absence of glands in this region.

clumped with surface secretion and bacteria. Areas with exfoliation of epithelium may be found, but is infrequent. Due to exposure and irritation to air and secretions, metaplasia of the epithelium is rather common, particularly when advanced polypoid changes have occurred and where drainage and ventilation is poor, as in the olfactory fissure and outer surface of the inferior and middle turbinates. It is very common to find that hyperplasia is frequently confused with metaplasia and so reported erroneously due perhaps to the fact that the rhinologist or pathologist is not thoroughly acquainted with the difference between the two. Ulceration of the epi-

thelial layer is very uncommon and even rare, but does occur more often in the hypertrophic suppurative type than that of the atrophic type.

b. Sinus epithelium differs essentially from the nasal type in that it is a simple columnar ciliated type, consisting: *1.* In the ethmoidal mucosa of columnar ciliated cells intermingled with olfactory and sustentacular or supporting cells. The olfactory cells lie between the sustentacular cells. Their nuclei are spherical, lie at different levels and more deeply placed than the sustentacular cells and form the zone of round cell nuclei, and *2.* in the antra, sphenoid and frontal sinus the mucosa is of simple columnar ciliated epithelial cells, many of which are of the secretory or goblet type. The goblet type predominate, particularly where hypertrophic and polypoid degenerative changes abound. Here we find them in very great number, replacing the columnar surface epithelium, where they seem to take on great secretory activity, due very likely to the presence of very few serous or mucous glands in the stroma or subepithelial layer. Hyperplasia of the epithelium in the ethmoid was not noted. In the antrum hyperplasia was rather common. Loss of cilia, or exfoliation or desquamation of epithelium in the sinuses is uncommon. Metaplasia is frequently found. Ulceration of the surface of the epithelium is rare and has not been noted in these studies of sinus epithelium except on one instance, although some authors do report their presence.

2. The subepithelial layer, hyaline layer or basement membrane, found in the mucous membrane of the nose and turbinates and rather faintly present, if at all, in the sinuses, is worthy of note. It is thought to be derived from the underlying connective tissue. It is composed of a dense but homogeneous layer of collagenous fibres, very faintly or totally unstainable on which the epithelial cells appear to rest, and through which spaces or canaliculi, migrating leukocytes are found working their way to the surface. It is always found in the mucosa of the nose as a rather distinct and sharp layer, and forms one of the differentiating points between nasal and sinus mucosa. It is occasionally found as a distinct layer in antral mucosa, but rare in the ethmoid, sphenoid or frontal. When present it is found associated with chronic inflammatory lesions, both of the hypertrophic and atrophic forms, as well

as in allergic disturbances, as asthma and allergic rhino sinusitis. In chronic inflammations, particularly of the atrophic form, it is found greatly thickened with epithelial exfoliation and seems to act as an important barrier or protecting agent against entrance of infection to the deeper layers of the mucosa. Some do not, however, consider it to have any clinical or pathological significance other than being an evidence of chronic epithelial irritation.

Fig. 3. Low power of antral mucosa in an allergic patient suffering with chronic antral suppuration. Note, cystic degeneration of mucous membrane; marked edema of stroma with diffuse infiltration of latter by cellular elements.

3. The tunica propria or stroma consists of loose connective tissue and blood vessels with infiltrating cellular elements, the latter varying in accordance with the degree and character of inflammatory changes present. In the nasal mucosa, particularly the turbinates, we must consider the glandular layer and cavernous layer. In the former is found many serous and mucous glands, and the latter consists of cavernous blood channels surrounded by a network of elastic tissue which enables the sinuses to fill and empty rapidly. The middle turbinate differs from the inferior in having less glands with predominating mucous glands, while in the inferior turbinate serous glands predominate. The middle turbinates have no cavernous blood spaces. Cystic degeneration of mucous

and serous glands is commonly found, particularly where polypoid changes are well advanced and presence of edema, causing a blocking of secretion in the ducts and the latter to become distended with muciginous material. The cysts assume frequently large proportions and become filled with cellular elements through invasion of a great number of pus cells. On reaching the surface, they rupture and empty their contents on the surface epithelium. The glands are lined by a single layer of cuboidal epithelium. When blocked up the cells secrete more actively and assume goblet cell structure.

The blood vessels are thickened and increase in number in accordance with degree of hyperplasia or hypertrophy. Proliferative changes of the layers are common, particularly the endothelium, which causing obstruction to the blood flow, increases stagnation and edema of the surrounding connective tissue. Thrombosis of blood vessels in this form of sinusitis is rarely if ever seen.

Cellular elements: Round cell or lymphocytic infiltration with polymorphonuclear cells is most common. Many eosinophiles and plasma cells are found, particularly in the allergic types of hypertrophy. The plasma cell is frequently mistaken for the eosinophile, and should, therefore, not be confused with one another.

Round cell infiltration varies in accordance with the amount of infection and local irritation. Areas of dense infiltration, both subepithelially and around gland structure, is very common, particularly in the chronic polypoid type, where suppuration is extensive. Areas where mucosa is densely infiltrated with cells to the point of pus formation is common, particularly in the advanced forms of antrum and ethmoid suppuration. Here cysts filled with pus and subepithelial collection of purulent secretion is common. Finck refers to the former as mesothelial cysts.

The antral tunica propria differs from that of the nose in the absence usually of a hyaline layer and presence of only a few secretory glands. Polypoid degeneration of the mucosa is very common with the predominant presence of cystic degeneration of glands. Marked round cells and leukocytic infiltration is also an outstanding feature.

The ethmoidal tunica propria has very few secretory glands. Here the secretory function is carried on by the epithelium, which assume goblet cell activity, secreting large quantities of muciginous secretion admixt with many lymphocytes and leukocytes that find their way to the surface through the basement layer.

The outstanding lesion of the tunica propria itself is usually a marked edema with resulting low grade minute polypoid or at times cystic degeneration with rarely polypi of any large size. The latter are rather rare within the ethmoidal laby-

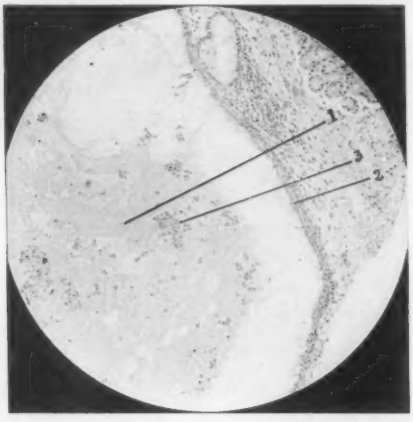

Fig. 4. High power of Fig. 3. Showing 1. Contents of cyst; 2. Cyst wall lined by epithelium; 3. Contents showing many leukocytes and mucus. Patient suffering with chronic suppurative sinusitis and vasomotor rhinitis.

rinth. Polypi coming from the ethmoid are usually polypoid degenerations of the nasal mucosa lining the ethmoidal or outer wall of the middle or superior meatus and rarely coming from the ethmoid mucosa.

However, with the presence of necrosis or resorption of the medial ethmoidal wall, small polypi from the ethmoid may extrude into the nose and assume large proportions, but this is rather rare. When the latter does occur they constitute the dangerous type of case, for in removing these polypi surgically one never knows where they come from. As it is possible that they may have their origin from the ethmoidal vault and in avulsing these polypi injury to the dura is pos-

sible. This type of case can be termed the dangerous type and when operated on frequently develop meningitis and terminate fatally. More will be said about this form of sinusitis when discussion of the bone pathology will be taken up.

4. The periosteum: The periosteum is derived from the connective tissue of stroma and is at times scarcely demonstrable. When present it may vary from a very finely defined bone covering to a markedly thickened and densely infiltrated structure, depending usually upon the degree of pathology present in the overlying tunica propria. The same cellular elements will be found infiltrating both the periosteum and tunica propria. Subperiosteal hemorrhage, both old and recent, are commonly seen and are frequently of traumatic origin. Edema, causing wide separation of the periosteal fibres, is frequently seen. Normally the periosteum in the ethmoid, sphenoid and frontal sinus is scarcely discernible so that unless the lesion is of long standing, one cannot definitely demarcate between the tunica propria and a periosteal layer, the former here taking the place and function of a periosteal layer.

5. Bone: The bone pathology constitutes the most interesting and most difficult phase in the pathology of chronic sinusitis. The pathologic bone changes met with are never of one or another distinct variety, but rather a mixture of various multiple pathologic processes going on simultaneously and one along side another.

The most frequent and common lesions noted were: *a.* bone rarefaction, resorption or atrophy; *b.* bone fibrosis; *c.* bone necrosis; *d.* bone hypertrophy.

a. Bone rarefaction, resorption or atrophy is most commonly seen. The process may be seen to begin as bone erosion, frequently due to chronic periostitis of the ethmoidal septa or the nasoantral wall, both middle and inferior meati. This process is evidently due to the presence of bacteria in or subperiosteally. This process of atrophy by erosion is rather slow and drawn out.

Bone atrophy is also very commonly seen associated with fibrosis and proliferative fibrous connective tissue changes. Connective tissue proliferation may be: *1.* endogenous, that

is, begin from within the bone spaces through proliferation of connective tissue about the blood vessels; and *2.* exogenous or connective tissue proliferation outside of the bone, *i.e.*, periosteum or stroma. Here primarily it starts with an erosion of the bone and subsequent invasion of bone by connective tissue. Thus thinning out of the bone takes place both from within and without, causing rapid bone resorption or atrophy. Fibrous tissue strands may be seen splitting the

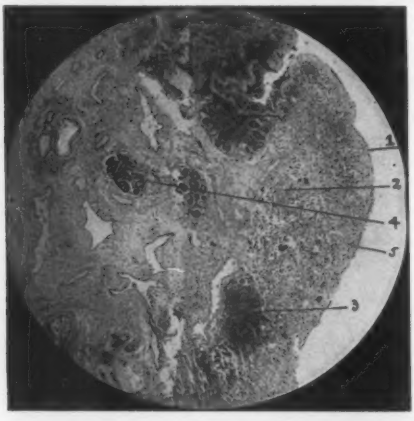

Fig. 5. Low power of middle turbinate mucosa in a patient having chronic suppurative pansinusitis with atrophy. Note, 1. Almost complete exfoliation of epithelial lining. 2. Marked cellular infiltration of stroma. 3. Crowding and atrophy of glands with periglandular infiltration of round cells. 4. Fibrosis of deeper layers with marked thickening of the outer coat of blood vessels and no evidence of thrombosis of latter. The hyaline layer is well defined.

bone into long separate longitudinal strands. Bridges of connective tissue may be seen separating islands of bone from one another. In case of the ethmoidal septa, they ultimately become entirely resorbed, converting the ethmoidal labyrinth into one or two large cavities. In case of the nasoantral wall, the latter is frequently converted into a thin fibromembraneous partition. Atrophic and fibrotic changes are commonly seen associated with extensive hypertrophies and polypoid degenerative changes in nasal structures, prominently in the middle and occasionally in the inferior turbinates. Here the

process is largely a mechanical one, of course associated with bacterial infection, etc. Endarteritis and vascular thrombosis, causing a shutting off of blood supply, is the chief cause of atrophy. The bone resorption here is very slow. The bone cells lose their sharp outline, appear pale and swollen, nuclei enlarged and pale, staining rather poorly at first and later not at all. The cells later fade and disappear, leaving empty spaces in the bone. The bone itself takes on a poor stain,

Fig. 6. Low power of mucous membrane of nasoethmoidal region (middle meatus) in a patient with atrophic sinusitis. Note, complete absence of epithelial lining; marked thickening of subepithelial layer (hyaline); presence of very few secretory glands and round cell infiltration about latter; marked fibrosis of stroma; very thick blood vessel walls with proliferative changes in endothelial lining of one large arteriole. No thrombosis, however, was found in any of the specimen removed from this patient.

looks pale and faded, gradually losing its outline until completely resorbed and its place taken by young connective tissue. This lesion is best studied in polypoid degenerative changes of the anterior half of the middle turbinate bone.

b. Bone fibrosis, as outlined above, is found in all advanced cases of chronic hypertrophic lesion associated particularly with suppuration. It is found associated with bone resorption, softening and atrophy. Also where pressure atrophy takes place due to polypoid hypertrophies, etc., fibrosis is present

in nasal pathologic lesions associated with infections, asthma and the so called allergies. Fibrotic changes are frequently met with in chronic ethmoiditis where complete resorption of the ethmoid capsule takes place, involving the ethmoidal vault and finally the dura of the anterior cranial fossae. The latter type constitutes the dangerous and frequent fatal cases when operated upon.

c. Bone necrosis is not as commonly seen as the above lesion just described and may even be considered as an infre-

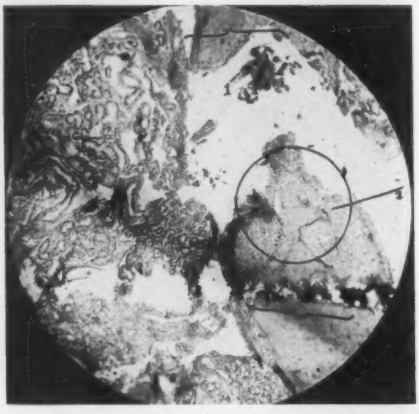

Fig. 7. Low power of bone necrosis of lower nasoantral wall in a young girl suffering with chronic suppuration of antra. Note, 1. Necrotic bone area between brackets with areas of devitalized bone and new connective tissue replacing dead bone. 2. Giant cell formation (osteoclasts) in Howship's lacunae.

quent finding. Necrosis was most frequently found in chronic antrum suppuration of long standing and involved chiefly the nasoantral wall. The lesion is considered as being caused by necrosis of the wall due to the involvement of perforating blood vessels, arteries or veins by an infected thrombus, the infection spreading through the mucosa lining the antrum, its wall and involving the nasal side. Although to a lesser extent necrosis is next in frequency seen involving the paper plate with exposure of the orbit and its contents to infection. When not disturbed by operative procedure the latter rarely takes place, although rupture of abscess pockets of the ethmoid into

the orbital cavity is occasionally seen. The floor of the frontal and anterior sphenoidal wall are other sites where necrosis occurs. The necrotic bone areas are eventually absorbed and replaced by fibrous connective tissue or new bone formation. In the latter the osteoclasts, multinucleated cells or the so called giant cells play an important role. They are found in the areas where bone repair goes on or in bony dugouts known as Howship's lacunae. It appears that due to irritation set up by infection, the osteoblast is capable of assuming the function both of the builder (osteoblast) and the destroyer

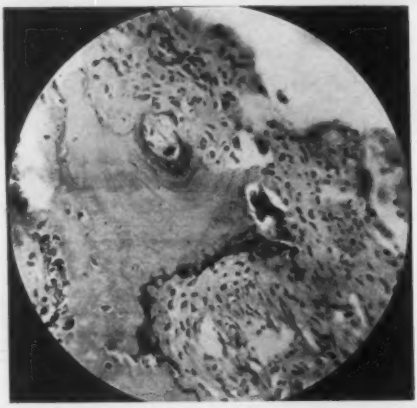

Fig. 8. High power of area in circle, Fig. 7, showing bone resorption with replacement by repairing connective tissue; marked osteoclastic activity and bone erosion; giant cells in Howship's lacunae; note how bone has become devitalized, staining poorly; bone cells also do not take the stain; nuclei are irregular and absent in most of the area.

of bone tissue (osteoclast). The osteoblast is said to be derived from either the periosteum or fibrous connective tissue of the tunica. The fibroblasts or young connective tissue cells lining the marrow cavities may also take on the function of both osteoblast and osteoclast. Von Gazer considers this metamorphosis of the osteoblast into the osteoclast as only functional and caused by the release of paraplastic substances from dying bone. Under normal conditions the osteoblasts appear as a line of dark stained, spindle shaped, singly nucleated cells, lined up end to end in regular order and parallel to the bone surface underneath the periosteum. Under de-

ranged or pathologic states they lose their clear and well defined outline, appearance and arrangement, standing up on edge, become irregularly enlarged, assume different forms and set to work as osteoclasts or bone destroyers, causing gradual erosion of bone surface by eating away the latter and ultimately absorbing the bone completely. Fibrous connective tissue ultimately displaces the bone as previously described under bone atrophy and resorption.

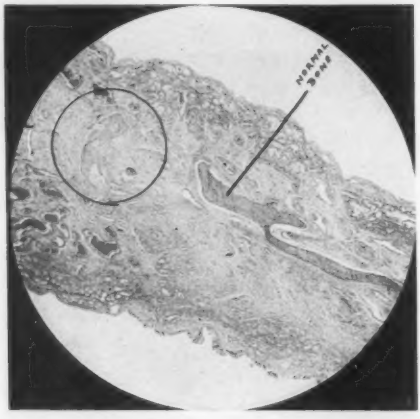

Fig. 9. Low power of middle turbinate tissue undergoing polypoid degeneration in a case of hyperplastic and polyypoid rhinosinusitis with asthma. Note, normal bone; area in circle showing bone resorption and marked thickening of blood vessels; fibrosis of stroma; loss of surface epithelium, thickening of subepithelial layer; atrophy of serous and mucous glands.

d. Bone hypertrophy is rather uncommon as a widely distributed lesion. It is, however, occasionally found in chronic hypertrophic lesion involving chiefly the inferior turbinate. The bony turbinal lamina of the inferior turbinate is normally thin and cribriform with the periosteum dipping down in these depressions and tightly adherent to them. Under chronic irritation and periostitis associated with chronic nasal suppuration and infection, the inferior turbinal bone (lamina) is found to be increased many times its normal thickness, frequently becoming so obstructive to respiration that nothing but a complete resection of this bone submucously will relieve the obstruction. Associated thickening of the adjoining sinus

walls are seen. This bony overgrowth is often of hard sclerotic character, making it exceedingly difficult to penetrate or resect the nasal walls of the antrum, sphenoid or frontal floor.

2. Atrophic sinusitis: a. Gross pathology. The gross pathology shows a decided roominess in the nose involving one or both nares, most frequently the latter. The roominess may vary from a moderate to marked degree where both nasal chambers appear exceedingly wide, exposing to direct inspec-

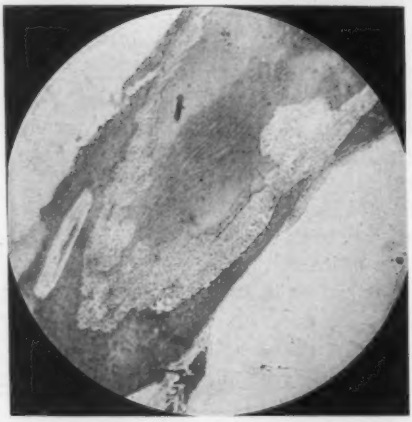

Fig. 10. High power of bone from nasoantral wall in a patient with chronic suppuration of antra. Note, bone erosion with fibrosis. A good example of endogenous and exogenous bone resorption with fibrous tissue replacing the bone; bone is pale and stains poorly; cells and nuclei diminished in number and absent in areas where advanced atrophy is present. Specimen was taken from lower antronasal wall.

tion the epipharyngeal wall and soft palate. In the advanced cases marked atrophy of the inferior and middle turbinates are present. When suppuration is present, pus, mucopus or crusts may be seen filling the nasal fossae and extending or dripping posteriorly into the nasopharynx. The mucosa of the middle turbinate is often covered with a thin, glazey looking secretion. During operation the middle turbinate is frequently found adherent to the nasoethmoidal mucosa (middle meatus) with low grade polypoid degenerative changes of mucosa between the two. Free pus is frequently found in this region, particularly on opening the ethmoidal cells. The

pharynx is atrophic, dry and glazy due to film of purulent secretions covering it. Crusts and thick pus may be found, extending down into the larynx. Extensive polypoid changes are never seen, the polypi often seen protruding from underneath the middle turbinate and ethmofrontal region are poor, impoverished looking specimen and never attain any large proportions.

b. Microscopic pathology: *1. Epithelial layer.* The epithelial layer of the nasal mucosa and turbinates shows usually a marked dissolution, exfoliation or complete absence of surface epithelium. In earlier cases the epithelial lining, although present, exhibits an unhealthy unevenness interspersed by areas of complete denudation, hyperplasia, metaplasia or clumping together of epithelium, cellular elements, bacteria and mucoid secretions. In advanced cases a complete loss of epithelium is most common. In the sinus epithelium degeneration and exfoliation is less marked than in the nasal chambers. Metaplasia with complete changes of columnar type of cell to the cuboidal or flat stratified and horny epithelial cell is quite common in the nasal mucosa, where it is exposed by roominess to swift currents of air and irritating substances, such as dust or fumes carried in by it. True ulceration of the surface epithelium, although occasionally met with, is considered rare.

2. Subepithelial or hyaline layer is usually prominently present and thickened, particularly when exfoliation of the surface epithelium is marked or entirely absent. Cellular infiltration, particularly of the round cell variety, is commonly seen diffusely infiltrating this layer and coming from the stroma below it.

3. Tunica propria: This layer, as in the hypertrophic type, consists of fibrous connective tissue frame work, glands, blood vessels and cellular elements infiltrating it. The general appearance of the stroma is that of a low grade chronic inflammatory process varying according to the length of time the condition has been present, and whether or not it has been superimposed by infection as well as to how recent the last attack of suppuration took place. Opinions vary among authors and pathologists regarding the relationship of simple atrophy to fibrosis and fibrous degenerative changes associated with ozena.

The common lesions found are round cell infiltration, in and around glandular tissue; glandular atrophy due largely to compression of surrounding fibrosis, the more advanced cases; localized areas of intense round cell infiltration; new blood vessels and fibrosis. Blood vessels are frequently seen to be markedly thickening, particularly in their outer coat. Perivascular lymphocytic (round cell) infiltration is commonly seen. Thrombosed blood vessels are rarely if ever seen. As a matter of fact, thrombosis of very small blood vessels was more commonly encountered in advanced hypertrophic rhinitis of the polypoid type and was found to be due to bacterial invasion of the lumen of the blood vessels rather than an endarteritis.

4. *The periosteum:* The periosteum is often thickened and infiltrated with the same cellular components involving the stroma next to it. In chronic and well advanced cases, bacterial stains show clumps of bacteria involving the periosteum or between the periosteum and bone.

5. *The bone:* The characteristic well defined atrophic cases of long standing, the skeletal structure of the nose and paranasal sinuses is that of a well defined subinvolution form characterized by retarded or underdevelopment of the nasal sinuses, associated with a diffuse sclerosis of its bony framework. The X-ray examination in these cases show a decided underdeveloped infantile type of sinuses in the older children as well as in the adults. The sinuses are small, poorly illuminated, with the frontals frequently absent. The ethmoids are sclerotic in type and very poorly developed and aerated when present. The outer nasal wall is markedly thickened and turbinal bony framework atrophic. The skeletal lesion in these cases is very much similar to the atrophy with ozena. Although occasionally one encounters advanced simple atrophy in children with large well developed sinuses, the reverse, however, is the rule. Microscopically, bone atrophy, sclerosis and bone hypertrophy are the predominating lesions.

235 East 22nd Street.

PRACTICAL APPLICATION OF VOICE DYNAMICS.*

DR. LEON FELDERMAN, Philadelphia.

The lecture this evening deals with voice dynamics in application to life and its intricate environment.

The dynamics of the human voice can be defined as a highly integrated activity of several complex structural groups which require extreme axiation, perfect coordination of the musculature involved. It is the potential power of the individual to utter either emotional sounds or articulate speech sounds. Articulate speech is the effector mechanism of voice production (dynamic effector design).

The mechanistic age has given us speech power in several states: acoustic, electric, mechanical, magnetic, optical and thermal. It would be well nigh impossible in this short space of time for me to enter into the special phases of speech development. Suffice it to say that I will limit myself to the normal behavior of the speech mechanism and treat the subject from the standpoint of physiology.

We have had various fantastic theories on the production of sound and it is not an overstatement to say that they are as prolific as cactus fields in Texas—and perhaps just as painful when mental digestion is expected. Lately, numerous teachers of music have entered the field of voice dynamics with their pet ideas on the speech mechanism. Not content, of course, to teach the subject, they went so far as to expound visionary ideas and have tried to explain the unexplainable. Not only have they been guilty of trespassing on ground foreign to their training, but have become so bold as to dominate the thought on the subject of human voice production. Unless we wish to have them invade us further and substitute confusion for clear thinking, as laryngologists it devolves upon us to join the physiologists and keep the subject away from speculative hands. We should extricate ourselves from the

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morass of loose thinking. We must help to standardize the study of the human voice, and those theories that do not conform to true physiological laws should be discarded to the limbo of exploded fallacies and be replaced by findings that will stand the test of the severest critics in our special field.

Darwin, in his book, "Origin of the Species," made references to vocal sounds in the lower animals. He spoke of the voice as an agency of defense or of combat, and as being largely influenced by the major emotions of life, such as love, jealousy or anger. It was Darwin who pointed out the swelling of the throat of stags during the breeding season. So, too, in the higher forms of life we note in the females during the period of gestation the thyroid gland takes on additional activity. There is overwhelming physiological proof that the internal secretion of the human body is strongly linked to the voice mechanism.

In the book, "The Human Voice, Its Care and Development," I speak of the Porichthys Natatus (California singing fish), which utters sounds. "This fish spawns at tidewater on the rocky shores of Monterey Bay during the months of June and July. At this time nests are made in cavities of large rocks and tide pools. There the fish, guarding their eggs, at the approach of anyone will emit a quivering or humming noise which does not lend itself to imitation. The sound of the paternal fish is produced by a mechanism of the air in the bladder which is responsible for a peculiar vocalization. The bladder of the Porichthys Natatus has developed by invagination from the pharynx comparatively early in embryological life."[1]

In bird life we have instances where the instinct of self-preservation for the young is demonstrated by the emitting of call sounds. The Stone period inhabitants resorted to pantomimic gestures of tongue and lips, comparable to deaf mutes of today, and these were reinforced by grunting sounds which helped to form primitive words and conveyed the thought of that day in its mysterious environment. Is it an atavistic urge in our make-up which prompts us to attend a pantomimic drama and go away satisfied?

There is a legend woven about Christopher Columbus when he discovered America, in his contact with the Indians. Facial grimaces and use of hands in gesture gave him the first ink-

ling of the Indian language which is so commonly referred to as the "Indian sign." It is interesting to recall the amusing comparison of the American doughboy during the late war, finding himself unable to comprehend the language of the French peasants and resorting to gesticulations which, though often grotesque, were especially effective when the sense of hunger dominated the situation.

When a voice sound is released from the mouth of a speaker to the ear of a listener, a pressure wave is created which is composed of a succession of condensations and rarefactions of the air. Such a wave follows the line of least resistance until it meets with solid objects; then a series of secondary waves take place. The pressure wave gives place to larger waves. In a large city, other things being equal, we can call to someone without losing much of the sound effect because the tall buildings encompass and localize the voice call. In an open field the same vocal note delivered at the same intensity and pitch would spread out and the sharpness of the voice be somewhat expanded and correspondingly diminished. Fundamentally, speeches are therefore composed of vowels and consonant sounds.

As you listen to me here this evening, there is a shifting of these sounds from one to another, but only one sound is heard at one time. When the sound stream is continued in a steady tone, it is said to be continuant, and when it is interrupted it is called a stop. A conversation, therefore, consists of continuants and stops.

We have in speech a comparatively steady state of vibration joined together in time, either by silences or transitions. Each of these states has its characteristic pitch and tone quality with its variations. In a case where a sentence is melodic or sung, the pitch changes are at definite intervals on the musical scale, with the consonants in subdued form, making it difficult for a listener to understand the meaning of the words. Singers aim to produce vowel quality and for this reason the more the vowel sounds are prolonged, the shorter the stop or friction consonants. The singer glides over the consonants but prolongs the vowels. Did this characteristic develop the opera? Perhaps its interpreters found the theme too foolish to speak, so decided to sing it instead to veil its absurdities.

Human sounds cannot be dissected like the human body. We rely entirely upon ear mechanism for a detailed description. Chemicals, on the other hand, are recognized by their atomic weight and other physical characteristics. The trained eye plays the role in its own classification. But in the world of sound we have only the stimulation of auditory apparatus contributing to the sum and substance of our sound experience. In the lower forms of animal life a highly specialized sense of smell, as in the ant, constitutes the speech organ; in some the optic nerve entirely guides the animal in his familiar environment, and in others it is the auditory nerve which interprets sounds beyond those of human responsivity.

SOUND.

A. Physical Phase.

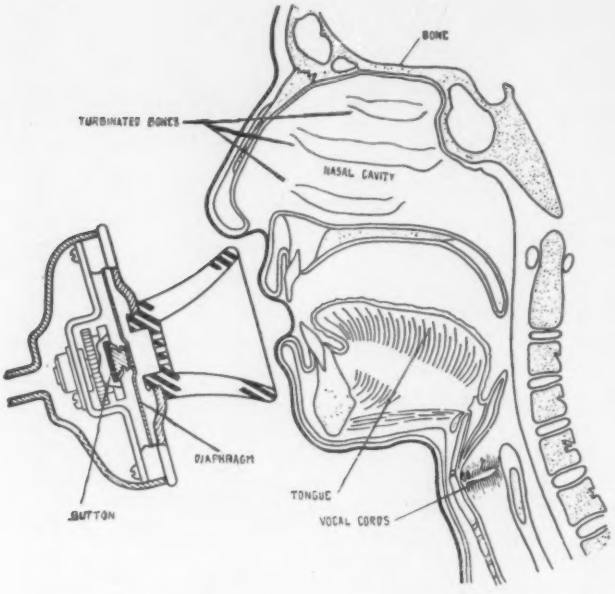
B. Experience Phase: 1. Sound experience or sound consciousness.

May I cite the following example to illustrate my point: An argument once took place between the followers of the physical phase and the followers of the experience phase. The former group cited the example of an island in the South Seas which contained a palm tree with cocoanuts, but otherwise uninhabited by a living being. One of the adherents of the "A" group remarked that if a cocoanut fell to the ground he believed that a resounding crash would take place when the cocoanut reaches the ground; whereas the adherents of the "B" class took an exception to this view, stating most emphatically that such could not take place since no living being was there to record the sounds.

Children first have a definite picture in their minds of the thought, and then proceed to express it in their own way. The speech center is a highly complex motor innervation, necessary to form appropriate sounds or words when certain concepts of thought are to be conveyed. Thoughts are not only expressed by the formation of intelligible sounds, but also by symbols.

Jean Baptiste Bouillaud, of Angouleme, was the first one to point out that aphasia is correlated with lesion in the anterior lobes of the brain. In 1825, Bouillaud[2] announced the so-called center for speech mechanism.

In 1861, Paul Broca,[3] the French scientist, precisely located the position of this important center in the posterior part of the third or inferior frontal convolution. The Broca's convolution or convolution center is synonymous with the center for speech.



Cross-Section of the Human Head and a Telephone Transmitter

Fig. 1.

The center of speech in right-handed people is usually in the left cerebral hemisphere. Aphasia in left-handed individuals indicates a lesion in the right side of the cerebral cortex. This has been confirmed by autopsies. Interference of speech, when it does occur either through disease or trauma, may affect the Broca's area, but may also extend to the contiguous brain territory, such as the middle portion of the Island of Reil and the opercular portion of the central convolution. Aphasia from whatever cause means loss of the power of speech.

An interesting form in which the patient's vision is perfectly all right, but his ability to understand written or printed words is completely lost, is called Alexia. The inability to make use of the lips or to write, although the patient can use his hands to draw, is called Agraphia.

Two forms of aphasia are recognized: *1.* Motor Aphasia; *2.* Sensory Aphasia.

In motor aphasia, the individual is unable to convey his thought. Pure motor aphasia is limited to the muscles of articulation. In motor aphasia there is inability to translate ideas or concepts into words. The patient is not dumb; there is no paralysis of his muscles of speech. Their ordered physiological cooperation, however, which is essential for normal speech, has become impossible for him; as, in childhood, he had to learn gradually how to use the organs of speech to form words, so, now, the kinesthetic memory pictures for the concepts mastered during the process of learning speech have become lost to him. "The distinction between this form of disturbance of speech and the anarthric is expressed most briefly and significantly in the French language: 'L'Aphasique ne sait plus parler, l'anarthrique ne peut plus parler.' "[4]

Sensory Aphasia—Wernicke: "Patient is unable to understand when spoken, or comprehend a written language, and spoken or printed symbols."

In sensory aphasia, the patient hears the spoken word perfectly, but can no longer understand it, as it no longer evokes in the consciousness its corresponding concepts. His mother tongue sounds to him as a foreign language, of which he has learnt little or nothing. He has lost his memory for the meaning of words. Sensory aphasia is often accompanied by paraphasia; that is to say, the patient makes continual mistakes in speaking and uses, instead of the correct words, other words of similar sound. This is explained by the fact that unconsciously we test, so to speak, internally the "ring" of each word before uttering it, by means of our sensory speech center. If this no longer functions normally, the patient very readily uses incorrect words, and is unable to recognize his mistake, as, in consequence of his sensory aphasia, he does not understand his own words.

A lesion of the speech center means that the motor mechanism of speech is intact. The lungs can ventilate the air freely. The vocal cords can still vibrate. The pharynx, tongue and lips can be moved as separate units, but curiously, the complex correlation of the motor functions of all these separate specialized functions is no longer possible.

Nerve impulses are not comparable to electrical current. Some physiologists have likened an impulse to a gun-powder fuse when lighted. The speed with which the fire travels down the fuse and the intensity of heat are in no way related; whereas loudness of tone registered in the ear depends entirely upon the active number of nerves excited, since each fibre is loaded down with its maximum impulse.

A nerve impulse, once it courses down the length of the nerve, meets with a refractory phase and it takes a greater degree of stimulation to bring a response, *e.g.*, fatigue. Should we apply an electric current to the leg of a frog, what happens? First, there will be a strong response, and if the electrical stimulant is repeated, the response of the frog's legs will be increasingly weaker, until there will be very little or no reaction, and one must send a greater current to make the same muscle respond. As the intensity of stimulus increases in the nerve trunk, the rate of nervous discharge correspondingly increases.

REFLEX TIME.

Considerable speculation has taken place over the question of reflex time in the speech mechanism. Thought generated in the cortical area immediately releases impulses which travel down the axons of Betz cells relayed to the corona radiata interior capsule, midbrain Pons Varolii to the neck; motor, sensory and sympathetic fibres participate in this tie-up. The motor nuclei 7, 10, 12 cranial nerves share in the proper distribution of these impulses through the musculature of the diaphragm, vocal cords, tongue, and lips.

Nerve centers involved in the reflex movement—the determination of the total time, which means the application of the stimulus and the beginning of the responses. It is the means we have of ascertaining the time required for the processes to complete a circuit.

H. von Helmholtz: The time required in the nerve centers was 12 times greater than that estimated for the conduction along the muscular and sensory nerves involved in the reflex. The stronger the stimulus, the shorter the time reflex. When the time reflex is involved the element of fatigue should be considered. Reflex time or reduced reflex time designates the time required for the processes to complete an action. Exner has estimated 0.0578-0.0062 seconds, equivalent to a wink of an eye.

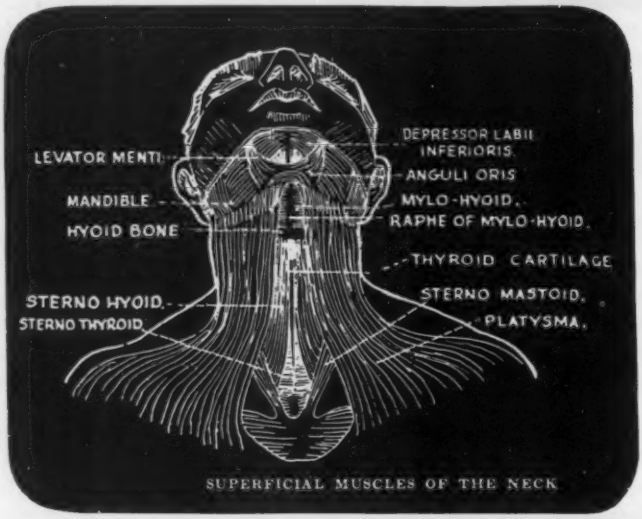


Fig. 2.

FORMATION OF VOWELS.

When confronted with the voice mechanism, whether in speaking or singing, the formation of vowels is of capital importance to the physiologist and the otolaryngologist alike. An avalanche of theories has fallen on our gullible heads which has added to our confusion rather than clarity of thought. It appears that any energetic student of the faculty of speech has woven a beautiful theory of his own, leaving us to wonder what next.

I shall not burden you with fanciful fantastic theories, but shall separate the wheat from the chaff and bring to light such dogmas as merit the attention of the followers of otolaryngology.

In the physiological world, the harmonic theory as first advocated by Wheatstone in 1837, still has close adherents today. According to this theory, the vocal cords generate a complex wave having a fundamental and a large number of harmonics. The component frequencies are all exact multiples of the fundamental. When these waves pass through the throat, the mouth and the nasal cavities, those frequencies near the resonant frequencies of these cavities are radiated into the air very much magnified, the amount depending upon the damping constant of the cavity. These reinforced frequency regions determine the vowel quality. Consonants are articulated with a great deal of energy and muscle force. The lips, teeth, tongue and hard palate help to form the consonants, particularly of the so-called fricative sounds.

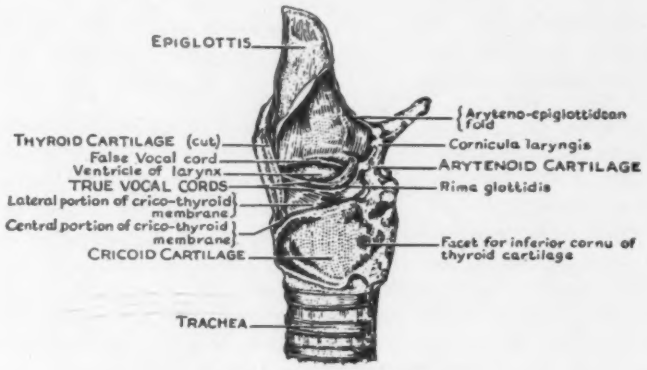
The inharmonic theory of Wills (1829) and Herman, and now advocated by Scripture, is growing more popular with the psychologists. The vocal cords act only as an agent for exciting the transient frequencies which are characteristic of the vocal cavities. In this inharmonic theory an invisible force sets these cavities into vibration. It has been said that a puff of air, passing through the glottis, is of sufficient strength and will set off the vocal cords into vibration. Just as soon as the vibrations occur, another puff of air replaces the preceding one. It is the succession of these waves which play upon the vocal cords with fair regularity that gives the speech mechanism its impetus.

As children we all resort to our own schemes of entertainment and diversion. I can recall, as a youngster, that I would go down along the banks of the Danube River in Rumania and playfully throw smooth stones with all the speed I could command, making sure they just skimmed the surface of the water, and then, to the delight of my playmates, would watch for the circular waves to develop. It was extraordinarily amusing to watch the circular waves develop so fast as to make a correct count impossible. In the beginning these rings would be very strong, the outlines very well defined, and in the course of a short space of time would become fainter and

fainter. Unless another stone were thrown in the same direction the ring waves would finally die out in the placid waters.

Not unlike the ripple in the quiet water, in the production of human sounds it is well nigh impossible to tell where the beginning is. We cannot very well name the exact location and we must, therefore, be satisfied with the acceptance of plausible facts.

Comparisons have been devised to serve those who wish to escape from the realities of life. It is a frank admission that the answer to the solution is another answer far more



Lateral view of interior of larynx.

Fig. 3.

evasive. It is answering a question by asking another bogus question. Comparison is a poor substitute. It is a limitless yardstick. For the greatest gift within human economy—the voice box—has withstood the asault of comparisons only to reveal to us that comparisons are not only odious but significantly valueless.

Savort, in 1825, compared the larynx to a whistle and took considerable pains to make it as realistic as possible. The larynx has been likened to a reed instrument, also to pipe organs. I suppose when we speak of physical mechanism for producing musical tones, we have the vibrating strings, *e.g.*, piano, violin or pipe organ. When we sound a note in any

of these musical instruments, we elicit more than one frequency of vibration. The lowest component frequency of vibration is called the fundamental and is usually determined by the pitch of the sound. A large number of component frequencies is called the harmonics. In recent years the mechanism of the larynx has been compared to an oscillator.

As we read books on the subject we will find that some authors speak of it as a vibrator in a purely mechanical sense. Among those that advocated the thought that the vocal cords swing like a pendulum, and that it is regulated by the oscillation of the vocal cords, are Helmholtz, Dodart, Biot, Liskovius, Longet, Mandl and Eustache.

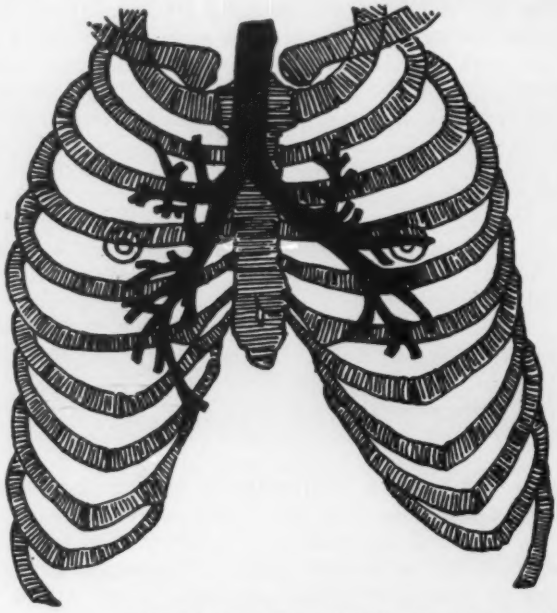
To vibrate, the vocal cords must be in a state of tension. Muller, Bataille, Fournie, Duval, Gay and Lermoyez have shared similar views.

Helmholtz, in 1865, stressed the *pharyngeal-buccal cavity theory* which constituted then the important phase in the chain of the vocal vibration. The vowel sounds are recognized through their high intensity and overtones. Both the French and German schools have given us enough theories to ponder and in America we have the opportunity to test these theories and accept only those which conform to the rational laws of physiology and physics. The German works of Ewald, Gutzmann and Musehold have given us the basic essentials of the mechanism of the larynx as a whole. The French works of Hellat (1898), Bonnier (1907) and Thooris (1927) have done a great deal to clarify some of the points in this apparently enigmatic study. We still have a number of physiologists who believe in the aerodynamic theory of which the inferior part of the vocal cords have an accessory part in the production of vowel sounds with or without the ventricles of Morgagni.

When one takes into consideration the rigid, cold, unyielding walls of the metal instruments, we can readily see that the best comparisons are poor. It is not a single factor which sets the vocal cords in vibration, but rather a combination of them. The air which passes through the inferior vocal cords is gradually released by the sub-glottic pressure which behaves more like an elastic sphincter and the human sound is initiated. The aperture of the glottis becomes narrower

to afford resistance to the intrapulmonic pressure. The vocal cords first become tense, and then undergo alteration in point of length because of its ability to give.

The larynx, with its nonlinear, irregular, soft, yielding musculature-like walls resembles only one organ and that is the larynx *per se*. It is futile to search our minds for a likeness of the larynx. You might as well look in a dark room for a black cat which isn't there.



RELATIONS OF BRONCHIAL TREE TO ANTERIOR THORACIC WALL, AS SHOWN BY X-RAYS. (BLAKE.)

Fig. 4.

Having thus far considered the theory of voice mechanism from the standpoint of fractional units, we are prepared to speak of the mechanism as one functioning group; that is to say, the machine as a whole. It is obvious that the ordinary process of the physiological function must not be inhibited if the human sound is to present a natural flow. Respiratory

tract and phonatory tract must work jointly and harmoniously in order to bring about perfect function. The cerebral, auditory, phonatory and respiratory tracts should function as a machine composed of one large unit.

Assuming that these tracts are normal and the singer wishes to phonate, or release a pianissimo tone, then a message from the Broca's center tells them to liberate a certain amount of energy to the muscles which help to phonate. Pitch intensity and breath pressure must be equal to each other, otherwise the individual will phonate off key. If he wishes to increase the force then additional muscles are called upon to do the work, and so till the singer brings all the muscles into play. In order to understand this we will consider some of the physical characteristics of the speech mechanism. In this group we have pitch, intensity, time, timbre and color.

Pitch is a physical property which brings the singer's voice within musical range. It directs and qualifies the singer. For example, if the singer is a basso, the pitch can reach as low as 32 double vibrations per second and can rise to 5000 double vibrations in the scale of music if the singer is a soprano. Naturally, it must be recalled that in the scheme of sounds the limits of audibility are from 16— to 20,000 double vibrations, but physiologically this becomes entirely too fine for differentiation and is completely lost on the ordinary human ear.

Intensity comes next in importance among the physical properties of the voice. In the world of sound no mathematical system can be applied to vocal phenomena which undergo alteration with each vowel or consonant utterance. A singing or speaking phrase depends a great deal on its intensity, which in turn relies on the subglottal pressure coming from the thorax. The following comparison illustrates this point: when a pitcher throws a ball, the act can be likened to pitch. The amount of energy back of the ball may be called intensity. In a painting, the ordinary picture, as of a tree, would be pitch, and the depth of the painting would be intensity.

Time is an essential factor in singing or speaking. It is what punctuation is to grammar. The element of time plays an important part in its relation to music, just as the drug

digitalis acts in relation to the muscles of the heart. I recall most keenly the long protracted marching of troops in which I participated during the World War. At the outset the troops marched with unity, their steps firm and springy. In spite of the heavy pack they had to carry, they would start off singing some popular song. It would generally be of quick tempo, such as "Over There." As the march continued and the soldiers became physically fatigued, their singing became less harmonious and lusty. They would then choose a song like "Mother, Dixie and Me," in which the tempo was slower. By the time the end of the march was reached, only a few in

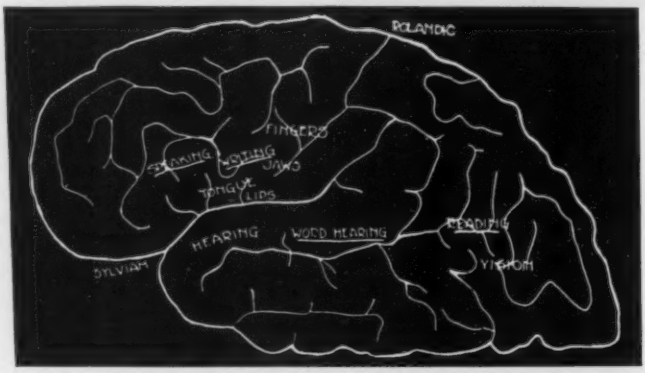


Fig. 5.

the battalion would have enough energy left to sing some sort of a song, and chronic fatigue could be detected in the still slower, weakened tempo of their voices.

Moreover, the tempo of a song often reveals the mental make-up of the composer. The autobiography of Chopin shows that when he composed his "March Funebre," the dirge-like content of the song with its melancholy feature was an integral part of his mental make-up.

What is known as color interpretation depends entirely upon the individual singer who must put the proper emotional effect into his songs. The play of emotions in a song contribute color, and this is in direct ratio to the emotional qualities of the singer.

Timbre is important in voice dynamics. The art of persuasion generated by the human larynx is definitely connected with the timbre, and the effective singer or speaker is governed by the timbre of his voice. This characteristic is so strong that it can often be traced back through several generations. Pathologically, other elements of speech, such as stuttering and stammering, may be changed, but the timbre make-up of a voice remains the same.

It is agreed that in ordinary conversational speech we use the upper register. The individual uses a minimum of breath pressure, and the vocal cords are held in tension while a certain amount of intrapulmonic air pressure is being released. The intrinsic muscles of the neck, such as the arytenoids, are used, and a certain amount of adduction takes place. This is true for singing as well as speaking purposes. When, however, the singer wishes to use the stronger muscles he calls upon the cricothyroids, and the intrapulmonic pressure is decidedly increased, while the lower register is called into action. The intensity is controlled by the proper use of the register. When one wishes to change from one register to another, the change is made at a low pitch. As the pitch increases the louder becomes the tone and the lower register is used. Sometimes the inexperienced singer will try to introduce the lower register at a high pitch, not allowing the arytenoids to assume the proper tension. Then there is a "mixed registration" and throatiness or, as the French call it, "coup de glotte." The avoidance of this embarrassing experience requires practice. To be able to keep the two registers apart requires intensive study. Incorrect registration robs the singer a great deal of his vocal range—in some cases it is fully half an octave, which makes all the difference between a good singer and the mediocre kind.

Teachers tell their pupils to relax completely. They often confuse fear with placement of tone. As you sit in your chairs this evening, from a physiological standpoint, you are not thoroughly relaxed. In every day life it is not possible to be absolutely relaxed; a certain amount of tension is necessary. So, too, in singing one must retain a certain degree of muscle tone. The good singer is never thoroughly relaxed despite the teachings of perfect relaxation, and singers who practice this form of singing soon pass into oblivion. For the only

class of people who enjoy thorough relaxation are those who have passed to the "Great Beyond." The old-fashioned cemetery with its permanent guests forms an admirable example of "perfect relaxation." The singer who has the faculty of choosing the middle road between relaxation and overtension is indeed fortunate. Otherwise, faulty registration results in faulty singing, and not infrequently the swelling of a note is associated with diminished tension.

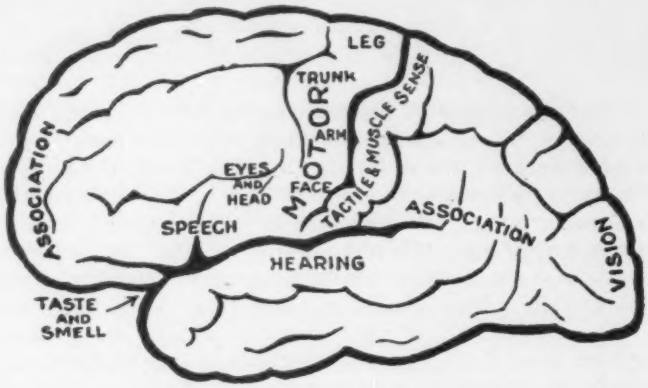


Fig. 6.

At the present time certain scientists are not in agreement about the resonance theory. Resonance is that property of the vocal mechanism which enriches the vocal tone. It draws its maximum from the vowel quality of the sounds, and a minimum from the consonants. When someone speaks to you over the telephone it is the resonance and the timbre of the voice which gives you the cue as to who is the party at the other end of the wire. Accurate resonating function is highly important in a singer, and the organs which participate in giving full resonance to the tone are divided into two classes: the adjustable cavities and the nonadjustable cavities. Among the former are the mouth, larynx (which is divided into oral, laryngopharynx and nasopharynx); in the latter we have the trachea, the bronchi, nose and sinuses.

Most of the singers who use poor quality in their tones are those who use their mouth exclusively as a resonator.

They are particularly handicapped because they have not learned to use the pharyngeal cavity as a resonating organ which would ensure a good, rich, melodic sound. The pharyngeal cavity has been more sinned against by omission than by commission; not by abuse, but rather by lack of use.

Napoleon, whose unmistakable wisdom and leadership in military life stands undisputed to this day, remarked: "God favors the army which is equipped with the heaviest artillery." At another time he is credited with having said that "the army marches on its belly." These axiomatic truths will always remain with us.

Today the morale of the troops is unduly stressed. During the recent carnage in the World War, the relief organizations, such as the Red Cross and the Y. M. C. A., have endeavored to sustain the morale of our youth with melodies. The popular songs were composed in simple cadence and filled with a flow of agreeable and ordered sounds. But above all they carried a message to the fighting troops. Tommy Atkins used to sing, "There's a Long, Long Trail A-winding," and the French poilu, hard pressed as he was by the German invasion, sang that song "Madelon," which was charged with a profound stimulant. It began: "Quand, Madelon vient nous servir a boire," etc. No matter how disheartened the French poilu might have been, even to the extent of dropping in his tracks from exhaustion, when the band struck up the tuneful melody of "Madelon," it struck a responsive chord and touched his patriotic heart so that he could carry on to a greater and more renewed activity.

It appears to me that the Frenchman, in that much abused phrase, "wine, women and song," was helped in the abstract to stiffen his morale, for in the abstract he could visualize "Madelon" serving him with that much-needed stimulant. In this country of ours we were not without popular melodies. The popular song which captured the imagination of our doughboy was naturally "Over There."

Adults can sing almost anything, but with children we should be more selective in our offering to them, particularly in the way of patriotic songs. It is on this subject that I wish to speak to you for a few moments.

When one analyzes the composition of these songs, we are struck by the great strain it puts on the immaturely developed child. A school child is at a tremendous disadvantage in singing a song to which he is not physically equal. The average school child today is not equipped with the necessary lung power, so he develops inhibitions and avoids the duty which we should try to make as pleasant as possible. The words of these songs are so difficult that in later years, when this child grows up, he hardly remembers the words. It is a mistake to overload the vocal tract of the child and bring about a loss of vocal equilibrium. The transition of one note to another is so greatly stepped up that the pitch of the voice breaks down and he develops the familiar "coup de glotte." This abrupt alternation of the pitch makes the child avoid a duty which he should tenderly embrace. I often wonder—since patriotic singing is so essential for good citizenship—why we have not composed a national song entirely suitable for children and another one for adults.

The human voice is a highly sensitive organ which governs sounds and is in turn governed by physiological laws. It is not true that one must inherit a highly cultured voice, for any voice can be improved through the proper application of the principles of speech or the technique of singing. It is equally untrue that one must have a supernormal larynx to bring about the desired effects.

For a speaker or singer to consummate properly his message to his audience, he should have a correct mental picture of his theme. The cerebral and auditory tracts must be perfectly intact. The respiratory actuator system should be in perfect accord with the vibrator. The larynx with the intrinsic and extrinsic muscles must harmonize. Only in this manner can we expect perfect axiation and coordination of voice dynamics.

REFERENCES.

1. Felderman, Leon: Human Voice, Its Care and Development.

2. Bouillaud Nouvells recherches sur le rhumatisme articulaire, etc., Paris, 1836. History of Medicine, Garrison, 3rd ed., page 433.

3. Bull. Soc. d'Anthrop. de Paris, 1861, II, 235-238, and Bull. Anat. de Paris, 1861, XXXVI, 330-398; History of Medicine, Garrison, 3rd ed., page 525.

4. Bing, Robert: Regional Diagnosis.

1327 Spruce Street.

AN UNUSUAL LOCATION OF AN ABERRANT THYROID.

DR. HYMAN SPORN and DR. HERMAN BOLKER, Brooklyn.

Aberrant and accessory thyroids are not unusual in the literature of recent years. When connected with the main body of the gland, they are called accessory; when completely separated, aberrant.

The usual locations, as classified by many authors, among them, Lahey, are:

A. Base of tongue — between hyoid bone and foramen cecum. *1*. Lingual — at foramen cecum. *2*. Intralingual — in the tongue. *3*. Sublingual — below the tongue. *4*. Perilaryngeal — in front of the larynx.

B. Within the superior mediastinum.

C. Just without the sternomastoid muscle, in the posterior triangle of the neck.

Our own case falls into none of these classes, being unusual in location and of origin difficult to explain. An extensive search of thyroid literature since its beginning in the nineteenth century fails to reveal a similar case.

Case Report: M. B., a white adult male, age 49 years, noted a swelling in the back of his head four years ago. The mass grew slowly but steadily larger until 16 months ago, when it was the size of a large hazel nut. No pain or discomfort were noted. At this time, the growth was excised, but never subjected to microscopic examination. A recurrence was noted three months later, rapidly reaching the original size. When seen by one of us (H. S.), the mass was located on the right side of the lower scalp, 3 cm. posterior and slightly below the mastoid tip. It was fairly firm in consistency, freely movable beneath the skin and over the bone beneath, and apparently part of the intervening tissue. There was no pain or tenderness.

A scar, 2.4 cm. in length, coursed vertically over the mass. General physical examination revealed no abnormalities. There was a right indirect inguinal hernia. The thyroid gland was barely palpable, and was not enlarged.

Under local anesthesia, the mass was again excised. Much free bleeding was encountered. No bands connecting it to deeper structures were noted.

Fig. 1. Low power to show architecture of thyroid tissue.

Pathological Examination (H. B.)— *Gross:* An ovoid, fairly firm and somewhat nodular, grayish-red mass of tissue, measuring 1.5 x 1 x 0.8 cm., partially covered by a thin fibrous capsule. On cut section, it consists of a rather homogeneous pink tissue with a few prominent blood vessels near the periphery.

Microscopic: Sections (see Figs. 1 and 2) are partially lined by a thin fibrous capsule from which a few trabeculae pass deeply into the glandular tissue. The majority of acini are small, of rather uniform size, lined by a single layer of cuboidal epithelium with pale oxytinic cytoplasm and central round or ovoid vesicular nuclei. There is no papillation into

the lumen. Colloid is present in approximately one-third of the acini; fragmented or vacuolated in the majority, with a slight tendency to take the basophilic stain in others. The fibrous stroma is fairly prominent and hyaline. There is scattered sparse lymphocytic infiltration; no follicles are present. The smaller blood vessels are congested. Basement membranes are intact; the acinar cells show basal polarity. There are no changes in the blood vessel walls.

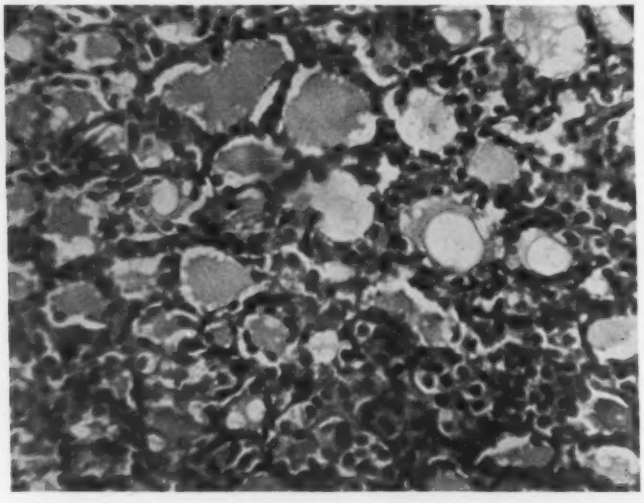


Fig. 2. High power to show detailed structure of thyroid tissue.

Various theories have been offered in explanation of these aberrant thyroid glands. They are either on an embryologic or pathologic basis.

Under the latter is mentioned the possibility of a localized adenomatous hyperplasia of the thyroid gland, regression and subsequent fibrosis, the last mentioned process producing a fibrous pedicle which is lengthened by tracheal and muscular movement with the final possibility of complete separation from the main body.

An embryological explanation has more often been sought. The thyroid anlage is a ventral median evagination (foramen

cecum) of the pharyngeal entoderm, just in front of the tracheal evagination. This first occurs in embryos of 3-5 mm. At first a hollow tube, its caudal solid portion grows down in front of the trachea, soon dividing into right and left lobes. The hollow tube (thyroglossal duct) is absorbed between the foramen cecum and the thyroid isthmus at the beginning of the fifth week. The supposed lateral anlagen from the fourth and fifth bronchial clefts have been definitely disproven by Grosser and shown to be the so-called ultimobranchial bodies, later incorporated in the adult thyroid gland.

The single median anlage explains the occurrence of midline aberrant thyroid tissue. In the case of the lateral aberrant bodies, however, difficulty is encountered. One suggestion has the thyroid tissue migrating laterally with the development of the tongue musculature, which originates from the branchial arch region. This does not explain our location posteriorly.

The normal histologic structure, lack of hyperplastic elements, and no marked vascularity takes this mass out of the class of the benign metastasizing strumas, as first described by Cohnheim and more recently emphasized by Simpson.

We offer as a possible explanation the downward migration of the original anlage, then secondary migration along the course of the occipital artery over the scalp posteriorly.

BIBLIOGRAPHY.

LAHEY, F. H.: *Surg. Clin. N. A.*, 4:1405, Dec., 1924.

GROSSER, O., KEIBEL and MALL: Human Embryology, 2:468, J. B. Lippincott, 1912.

MARINE, D.: Cowdry, Special Cytology, 1:551, Paul B. Hoeber, 1928.

GROSSER, O.: *Anat., Anz.*, 37:337.

762 St. Marks Avenue.
861 Park Place.

CURED CARCINOMA OF NOSE AND ANTRUM.

DR. MILTON H. SHUTES, Oakland, Calif.

Examination of indices of recent medical literature revealed no report of cured intranasal or maxillary sinus malignancies. In the *Annals of Otology, Rhinology and Laryngology*, December, 1932, Dr. Joseph H. Bryan of Washington, D. C., reported a case of papillary carcinoma of the maxillary sinus that after six months' observation, showed no apparent signs of return; and he concluded his report with the following sentence: "Owing to the intensive work that is being done on cancer all over the world, I believe all cases treated with X-ray and radium should be recorded."

On the basis of the generally accepted opinion that a case of treated cancer which shows no evidence of recurrence after five years may be considered as cured, the following case was presented before the regular staff meeting of The Samuel Merritt Hospital, Oakland, California, on February 6, 1933, the fifth anniversary of the operation.

In January, 1926, Mrs. C. B. L., age 51 years, complained of a left tinnitus auris. Mild chronic catarrhal rhinitis, no intranasal obstruction, the Roentgenograms showed cloudy antra; no surgical interference. The tinnitus subsided after six weeks of treatment.

Two years later the patient returned complaining that the left side of her nose had been stopped up for four months, and of a sense of pressure and swelling over her left cheek. The left side of her nose was completely filled with a cauliflower-like mass which bled on manipulation. Biopsy diagnosis by Dr. R. A. Glenn was, "papillary squamous cell carcinoma, grade II." Feb. 6, 1928, under general anesthetic, the usual incision for the Caldwell-Luc operation was made. The underlying bone was soft and the only instrument needed was a curette. The contents of the antrum were poured and scooped out—soft, running mass of pus, blood and epithelial masses. The bleeding was sufficiently controlled to permit

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the use of the desiccation current. The antral walls were thoroughly bombarded and made as dry and crisp as overdone toast. Then the intranasal mass was transfixed a number of times for 5 or 6 seconds with a needle and thoroughly cooked by means of coagulation. Patient left the hospital in good condition the following day.

From Feb. 7 to 13, six high voltage X-ray treatments were given over the nose and left antrum, totalling 1350 r-units, with ½ mm. copper and 1 mm. aluminum filtration.

Feb. 14, much sloughing from left nostril and antrum. Able to breathe through left nostril after saline irrigation.

Feb. 17, antrum clear, nose quite free and ready for radium.

Feb. 18, 500 mgr. hours of radium with 1 mm. brass and 2 mm. rubber filtration in the left nostril, followed by 750 mgr. hours of radium with ½ mm. lead and 2 mm. rubber filtration in the left antrum.

March 2, nose and antrum fairly clean; still some sloughing.

March 21, much improved.

May 18, nose and antrum clean except for a 10 mm. area on the floor of the antra ½ inch back of surgical opening.

July 10, the suspicious area near alveolar opening still present, so 1200 mgr. hours of radium was given with ½ mm. platinum and 2 mm. rubber filter. The pack was placed in the center of the antrum and surrounded by gauze.

Sept. 1, the area in question only 2 by 3 mm. in diameter.

Oct. 13, nose and antrum free of pathology.

April 11, 1929, no evidence of return of carcinoma.

April 24, 1930, no evidence of return of carcinoma.

July 28, 1931, no evidence of return of carcinoma.

Sept. 2, 1932, no evidence of return of carcinoma.

Feb. 6, 1933, five year cure.

NOTE: Since the above was written, Dr. G. Allen Robinson, New York, in THE LARYNGOSCOPE, April, 1933, reported 10 cases of carcinoma of the maxillary antrum as having "good results" after periods ranging from 2 to 10 years.

501 Wakefield.

p 76

RESEARCH STUDY CLUB OF LOS ANGELES.

The Research Study Club of Los Angeles will present its fourth annual clinical course from Jan. 21 to Feb. 1, 1935, in Los Angeles. The guest lecturers in Otolaryngology will be Dr. George Portmann, of Bordeaux, France, and Dr. John Barnhill, of Indianapolis and Miami. The guest lecturers in Ophthalmology will be Dr. Webb Weeks, of New York, and Dr. Harry Gradle, of Chicago.

As usual, the ophthalmological subjects will be given in the forenoon, while the otolaryngology will occupy the afternoon and evenings. The courses in dissection, given by Dr. John Barnhill, will be given in conjunction with the University of Southern California Medical School. The fee for the entire course will be $50.00.

For further information and a detailed program of the course, please communicate with Dr. Don Dryer, 2007 Wilshire boulevard, Los Angeles.

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